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THE OTOLARYNGOLOGIC ASPECTS OF SKIN AND SCUBA DIVING.*

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"Man is essentially a terrestrial being, and his anatomy and physiology are not modified for a water environment. When man is out of his normal sphere, he must understand what limitations nature has placed on him and not ignore the fundamental laws that regulate his own being."

This observation was made by Taylor[52] in 1939, and for many years stimulated his study of biology and comparative physiology as related to otolaryngology. Though man lacks appropriate adaptation to an aquatic environment, he has found other means of protecting himself in such an environment. Taylor[53] demonstrated that man has adapted himself to surface swimming by proper methods of breathing which protect his respiratory epithelium from the ill effects of water, and later Taylor and Dyrenforth[54] reported research which established that the prevention of chilling with its associated lowered resistance and subsequent infection is possible through time limitations and exercise in the water.

In recent years, man has become restless with his surface existence and through his ingenious ways has devised methods of exploring the fascinating worlds of the atmosphere above and the sea below. His ultimate future in these two environments will probably far exceed the concepts of the keenest

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imaginative minds of today. The modern physician must assist his fellow man in these endeavors and familiarize himself with the physics and physiology of the subaquatic and space environments. The military physician has made a great contribution in this field, and without his understanding and guidance man's progress in these two realms would be very limited.

Sports diving has today become a recreation for millions of people throughout the world. The underwater diver, equipped with the snorkel (underwater breathing tube), face mask, and flippers is commonly known as a skin diver. The time and depth of his underwater explorations are dependent upon the length of time he is able to hold his breath. In 1944, a French naval officer, Capt. Jacques-Yves Cousteau, and a French engineer, Emile Gagnan, designed the Aqua-Lung. This device consists of a tank or tanks containing air under pressure which delivers the air through a tube and mouthpiece to the swimmer at a pressure comparable to that of the aquatic depth he has attained. The equipment is commonly known as self-contained underwater breathing apparatus, conventionally shortened to the term "scuba." The duration of the underwater period and the aquatic depth the diver attains with this equipment are limited only by the physiology and physics which govern his being and the amount of air in the tank. In addition to protecting his respiratory tract from the water and preventing body chilling, he must now adapt himself to the effect of the pressure exerted on his body by the subaquatic environment.

Every physician should know the medical problems and dangers inherent in skin and scuba diving. This knowledge is of the essence to the physician who resides near water which attracts the curious of all ages. There are avid enthusiasts in every state in the Union. Wherever there is an old water-filled quarry of sufficient depth to invite curiosity, there will probably also be a group of these divers.

Many of the medical problems involved are related to the specialty of otolaryngology. Otolaryngologists should be cognizant of these problems, both from the standpoint of prevention and of treatment. They should be prepared to offer

advice as to the physical requirements for this sport and should discourage those persons from diving who are not physically or mentally fitted to engage in it.

DIVING PHYSICS.

For practical purposes water is incompressible. Air is easily compressible, and changes in volume resulting from small changes in pressure are readily observed. Atmospheric pressure is due to the weight of air just as water pressure is due to the weight of water. An immersed body is subjected to the sum of the atmospheric and water pressures. This value is known as absolute pressure. Atmospheric pressure at sea level is the weight of a column of air one square inch cross section having a length extending in the vertical direc-

Depth in Feet	Atmospheres	Pounds Per Square Inch
0	1	15
33	2	30
66	3	45
99	4	60

tion as far as the atmosphere extends. This measured pressure is approximately 14.7 pounds per square inch, and it is called a pressure of one atmosphere.

One atmosphere of pressure at sea level is equivalent to a one inch square column of water 33 feet high and beneath the surface. Every foot of depth of sea water produces a pressure of 0.445 pounds per square inch, and every 33 feet of sea water exerts a pressure equivalent to one atmosphere of pressure. The atmosphere above the surface is also exerting a pressure of 14.7 pounds per square inch.

According to Boyle's law, the volume of a body of gas varies inversely to the pressure to which it is subjected if the temperature remains constant. The density, however, varies directly. Fig. 1 illustrates the effect of pressure on the lung of the skin diver and Fig. 2 demonstrates the equalization of pressure on the scuba diver.

Since the human body is composed mostly of solids and

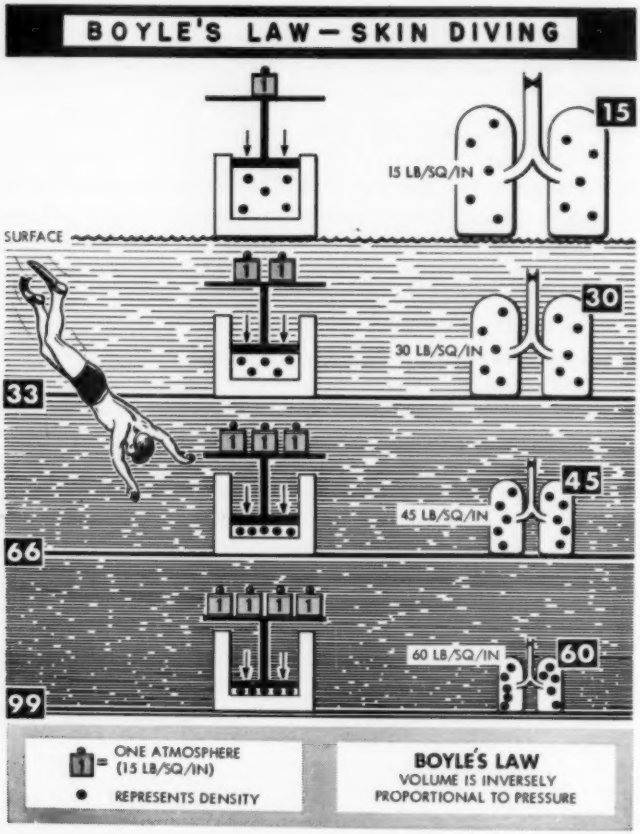

Fig. 1. Boyle's Law—Skin Diving. Pressure changes occurring in the lung of the skin diver with the glottis closed. The lung is compressed by the external ambient pressure.

A. At 33 feet (2 atmospheres absolute pressure) the pressure is doubled, and the volume is half.

B. At 66 feet (3 atmospheres of pressure) the volume is one-third, and the pressure tripled.

C. At 99 feet (4 atmospheres) the volume is one-fourth, and the pressure is increased four times.

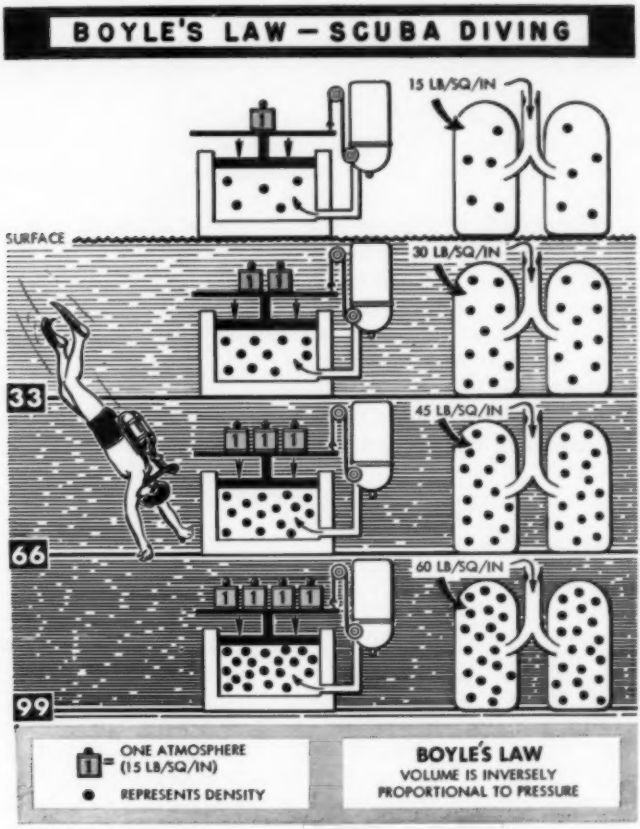

Fig. 2. Boyle's Law—Scuba Diving. The scuba delivers air through the open glottis to the lung at a pressure that varies with the ambient pressure.

A. At 33 feet (2 atmospheres absolute pressure) the lung volume remains the same. Pressure and density are doubled.

B. At 66 feet (3 atmospheres) the lung volume remains the same. Pressure and density are tripled.

C. At 99 feet (4 atmospheres) the lung volume remains the same. Pressure and density are increased four times.

fluids, it is able to withstand increased pressure well. Behnke,[5] in 1944, observed that the remarkable phenomenon of adaptation to environmental stress is exemplified by man's tolerance to rapid and extreme alterations in barometric pressure. "If pressure alone is considered," he stated, "apart from the effect of gases in the viscera, in the aural and sinal spaces and in solution in tissues, then variations in pressure in the range of 0.11 atmospheres (50,000 altitude) to 16.1 atmospheres (500 feet diving depth) are without physiologic effect." Pressure differentials developing from localized inability to transmit the increased pressure lead to pain and disease in the skin and scuba diver. This effect takes place mainly in the middle ear and sinuses. The scope of this paper is limited to the problems of the ears and sinuses and the relationship of the larynx to the development of air embolism, pneumothorax and mediastinal emphysema. The numerous problems of diving as related to the effects of the partial pressures, and solution and evolution of gases are not included.

OTOLARYNGOLOGIC PROBLEMS.

Pressure Factors.

The normal ear maintains a pressure equal to that of the immediate environment. The eustachian tube provides the only means of equalizing the air pressure in the middle ear to the changing pressures of ascent and descent. The main function of this tube is to ventilate and drain the middle ear. Its pharyngeal ostium is normally closed except when opened by positive pressure in the nasopharynx or muscular activation by the tensor veli palatini, levator palati and salpingopharyngeus muscles. The nasopharyngeal ostium of the tube acts as a valve which is normally closed. The respiratory ciliated epithelium beats downward toward the nasopharynx. A positive pressure in the middle ear opens the eustachian tube with little difficulty, as occurs in ascent.

Armstrong and Heim[3] demonstrated that in descent the eustachian tube acts as a flutter valve and remains closed under all degrees of pressure unless it is opened by voluntary action. One subject tested to a pressure of minus 470 mm. of mercury suffered a tympanic perforation. These authors

showed that voluntary opening of the tube will immediately equalize pressure completely. When a negative pressure of from 80 to 90 mm. of mercury or more had developed in the tympanic cavity, it was impossible to open the tube voluntarily. This degree of negative pressure held the fibrocartilaginous portion of the eustachian tube collapsed. The atmospheric pressure then had to be decreased before the tube could be voluntarily opened.

Aero-otitis Media.

A pressure differential between the air of the middle ear and the external environment sets up a pathological state in the middle ear. Armstrong and Heim[3] designated this condition aero-otitis media and stated that it commonly occurs during change of altitude in airplane flights. It is characterized by congestion, inflammation, discomfort, and pain in the middle ear, and there may be tinnitus, vertigo and temporary or permanent impairment of hearing. Bleeding may occur in severe cases. The pathological changes may, in fact, vary from slight congestion to extensive rupture of tissue. Aero-otitis media is probably the most common pathological condition resulting from flying and skin and scuba diving.

In the person with a widely patent eustachian tube aero-otitis media would not develop on ascent or descent as there would be a free flow of air between the middle ear and the nasopharynx. Any condition which enhances the narrowing of the tube increases the likelihood of aero-otitis media's developing.

The degree of pathologic change caused by otitic barotrauma varies with the amount of pressure differential between the middle ear and the ambient pressure, and the length of time before the pressures are equalized. The tissue changes are due to a suction effect, the mechanism of which might be compared to that which occurs when the suction bulb is put on the arm in the capillary fragility test.[15] In the mild case of barotrauma there is a slight retraction of the tympanic membrane and a dilatation of the malleolar vessels and the vessels in Shrapnell's membrane. The vessels in the middle ear also expand, and the negative pressure causes them to

become more dilated and engorged. In prolonged and severe barotrauma there is an increased engorgement of these vessels and more pronounced retraction of the tympanic membrane.

Small subepithelial hemorrhages may occur in the tympanic membrane. The mucous membrane of the middle ear becomes congested and swollen. The eustachian tube becomes in-

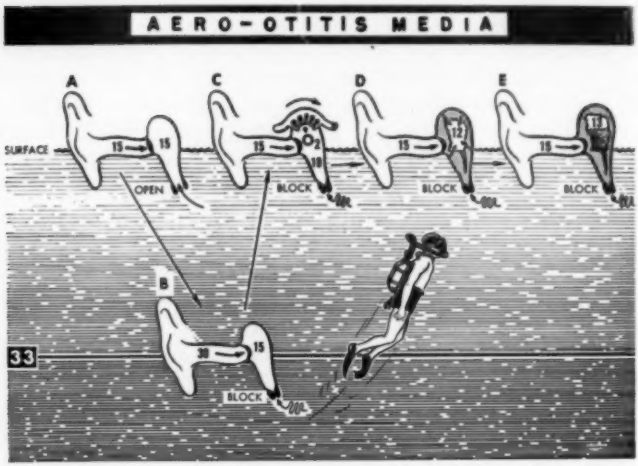

Fig. 3. Aero-otitis Media.

A. The eustachian tube is open. The ambient and middle ear pressures are equal.

B. With descent the pressure differential causes barotrauma, and the eustachian tube becomes blocked.

C. The eustachian tube remains blocked as the diver ascends because of pain. The negative pressure in the middle ear is increased at the surface by absorption of oxygen from the middle ear.

D. Edema of the mucous membranes and decreased volume of the middle ear. Decrease in volume leads to decrease in negative pressure.

E. Serosanguineous fluid forms in the middle ear and decreases the volume of the middle ear further. Decrease of volume tends to decrease in negative pressure.

creasingly swollen and blocked and frequently closes off the tympanic cavity. Absorption of air by the vessels in the middle ear enhances the vacuum, and an increasing engorgement of the vessels and edema of the membrane follow. A transudate of serous and serosanguineous fluid forms. If the negative pressure is sufficient, there may be a rupture of

vessels in the middle ear and the cavity fills with blood. Sufficient fluid forms to equalize the ambient pressure. In very severe barotrauma a rupture of the tympanic membrane occurs (see Fig. 3).

Aero-otitis Media and Hearing Loss.

Excellent studies have been carried out by the Navy and other military services to determine the relationship between barotrauma and hearing. Some of the most thorough studies have been made at the submarine escape training tank at the U. S. Naval Submarine Base in New London, Conn. Candidates for the submarine service are subjected to 50 pounds of increased air pressure in a recompression chamber prior to entering the water for actual training with the submarine escape appliance. As pressure is increased in the recompression chamber, an inequality of pressure from the two sides of the tympanic membrane is created because of the restricted flow of air through the narrow eustachian tube. The majority of men are able to equalize the pressure in the middle ear as pressure is increased through swallowing, yawning, or Valsalva's maneuver. In some cases no maneuver is effective in bringing about the equalization of pressure, and if the pressure is increased, aero-otitis media develops.

Etiological factors, mentioned by Armstrong and Heim,[3] which may prevent ventilation of the eustachian tube under pressure were: recurrent acute respiratory infections causing blockage due to an inflammation of the mucosa and lymphoid tissue in the tube, nasal obstruction, sinusitis, tonsillitis, tumors of the nose and nasopharynx, paralysis of the soft palate or superior pharyngeal muscles, enlargement of the pharyngeal or tubal tonsil, inflammatory conditions of the eustachian tube or middle ear, and scar tissue about the ostium of the eustachian tube. If the tube is blocked, the increase in pressure must be stopped or there will develop a sharp pain in the ear and damage to the tympanic membrane or middle ear.

In addition to the etiological factors listed by these authors, one must consider anatomical narrowing of the eustachian tube. In my case, reported herein, most of the difficulty was in the right ear. Examination of the nasopharynx revealed

no difference on the two sides. One of the divers in my series was able to dive 80 feet, by skin diving, without difficulty with his ears. He had no trouble in equalizing pressure unless he had a severe cold. On examination, it was possible to see him move the tympanic membrane back and forth by merely breathing in and out through the nose. Great variation in the ability to ventilate the tube may in such instances be due to the anatomical factor of a rather wide tube in one and a very narrow tube in the other. Another diver was able to move the tympanic membranes back and forth by the to-and-fro movement of the jaws. This effect suggests that malocclusion might have some bearing on the ability to equalize pressure in the eustachian tube.

In a study by Schilling and Everley,[50] 152 of 2,751 men subjected to 50 pounds of pressure in a recompression chamber experienced aural symptoms so severe that they had to be decompressed and removed from the pressure chamber, usually before the pressure reached 10 pounds. The most frequent and the most serious complication of aero-otitis media was the associated loss of auditory acuity, which was at times sufficiently great to be disabling. In favorable cases it took a month for the hearing to clear, and in some cases it did not return to normal. The hearing loss was of the conductive type. As to therapy, these authors concluded that time was the only treatment available since it was necessary to await the organization and final absorption of the blood and serum in both the tympanic membrane and the inner ear. In addition, they used ear drops and ephedrine nasal spray. They believed that permanent damage to hearing, particularly in the higher frequencies, may result in any ear in which damage to the middle ear has been extensive. They also concluded that taking of pressure is not the cause of loss of auditory acuity, but that the loss is associated with damage to the middle ear, caused by inability to equalize the air pressure. They regarded compressed air illness as a rare cause of loss of auditory acuity.

Teed's classification of the otoscopic findings after pressure tests has been widely accepted: Grade 0, a normal-appearing tympanic membrane; grade I, a retraction of the tympanic

membrane with redness in Shrapnell's membrane and along the manubrium; grade II, a retraction with reddening of the entire tympanic membrane; grade III, the same as grade II except that fluid is present in the middle ear; and grade IV, hemotympanum or perforation of the tympanic membrane, or both. Teed[55] reported that in his studies hearing loss was encountered in all grades except 0. In grades I and II hearing was restored within a few hours, but in grades III and IV, weeks or even months were required for recovery. In a few cases the hearing loss was permanent.

Teed[55,56] noted that the longer a man stayed under pressure after pain began, the higher was the grade of aural damage. Those who were able to equalize up to 12 or more pounds of pressure and then experienced pain were, in general, found to have stopped equalizing and after noting pain, were no longer able to equalize the pressure.

Relief from tubal obstruction should not be expected as a results of tonsillectomy or submucous resection, in Teed's opinion. Spontaneous recovery of damage to the ears was the rule in the large series studied. This author observed that grade III ears, if not cleared in a week, may need a myringotomy.[55] He advised against ear drops and stated that nothing should be placed in the ear with a perforated tympanic membrane. His conclusion was that only adenoid tissue in the tube, upper respiratory infection, and possibly allergy are important as etiologic factors in the causation of obstruction of the eustachian tube. He observed that contrary to the belief that repeated autoinflation in the presence of an upper respiratory infection would cause otitis media, this disease developed from this cause in only one of 30,000 men, many of whom had acute upper respiratory infection at the time of the study. In his opinion, the otitis media probably results from pressure changes associated with tubal obstruction and not from autoinflation. Hearing loss depends on the degree and duration of the pressure differential and the degree of pathologic change sustained secondary to the barotrauma.

Haines and Harris[21] evaluated the responses of 6,149 men at 50 pounds of pressure (3.4 atmospheres) for from three to

ten minutes. Of this number, 26.9 per cent contracted aero-otitis media. They observed that a feeling of fullness or congestion in the ear following pressure may be confused with loss of hearing. Many of the men in their series with this complaint had normal audiograms. They noted almost no effect on auditory acuity after exposure to pressure except for middle ears filled with blood unmixed with air, and concluded that the deafness was a result of dampening of the ossicles by the fluid in the middle ear. This type of ear they classified as grade V, adding one category to Teed's classification, the only difference from grade IV in that classification being the presence of free serosanguineous fluid without entrapped air. For the four types of ears classified by Teed[55] they noted little change in the acuity of hearing.

Their studies indicated that the patient having difficulty equalizing eustachian tubes under pressure will continue to do so unless some mechanical alteration takes place in the mechanism of the eustachian tube. The local use of Neo-Synephrine was of little value in their series, and no improvement resulted from psychotherapy. Ninety per cent of the patients treated with radium to reduce the size of the hyperplastic lymphoid tissue in or about the tube became able to sustain pressure without aero-otitis media's developing. Dental treatment was also undertaken to realign muscles to the eustachian tube by alleviating improper motion of the jaw.

These authors observed that in a good percentage of the men in their series aero-otitis media developed for no obvious reason when they were under pressure. They believed improper inflation was responsible because the men, although capable of autoinflation, would let the increasing pressure "get ahead" of them one or more times and then would inflate the ears only after some damage had been done.

In evaluating the effect of barometric pressure changes on hearing, Kos[29] concluded that aviators may have repeated attacks of aero-otitis media without sustaining a permanent loss of hearing. He believed that few Air Force combat personnel suffer aural disability as a result of pressure differentials associated with flying.

The use of irradiation to the nasopharynx is being advocated for those having repeated bouts of aero-otitis media as a result of engaging in diving either as a vocation or an avocation. Duffner[15] recently proposed that the monel metal radium applicator containing 50 mg. of radium be applied three to eight times to the pharyngeal orifice of each eustachian tube for a period of eight to ten minutes at intervals of two weeks to one month. In my opinion, radium should not be applied to the nasopharynx without a full understanding of its potential dangers. The patient should be advised of the inherent and unknown dangers of this therapy. Otolaryngologists should not take lightly the admonition of Lampe[30]: "On the basis of the known biologic effect of Beta radiation, the available data on the magnitude of the doses (as expressed in Roentgens) which are currently used, and the knowledge that the approved technique of repeated irradiation enhances radiation damage, the conclusion appears inescapable that permanent and significant radiation damage to the nasopharyngeal soft tissue is being produced not infrequently. This being the case, one can anticipate encouraging an unpredictable incidence of the clinical manifestations of late radiation complications at some future time which may vary from a few to many years after treatment." I would not advise the use of the radium applicator for a sports diver; instead, I would advise that he seek another avocation. Should I recommend it to the professional diver, I would inform him of its potential dangers.

Two cases involving impairment of hearing as an occupational hazard associated with diving are presented.

Case 1. In March, 1958, I examined F. McC., a 36-year-old man, who had been employed as a diver for 12 years at Marine Studios, Marineland, Fla. He complained of a hearing loss of eight weeks' duration. Wearing a rubberized canvas suit, the divers at Marine Studios dive in a circular tank 12 feet deep and a rectangular tank from 12 to 18 feet in depth. They wear a helmet weighing approximately 80 pounds, which delivers 25 pounds of compressed air to the diver throughout the dive. The diver usually opens the eustachian tubes by swallowing or movement of the jaws. If necessary, he may put his hand up under the helmet, hold his nose and carry out a Valsalva procedure. The duties of this patient required that he dive five or six times throughout the day to clean the tanks and feed the fish during the various shows. There was a history of pain in the ears three or four years previously while he was diving in 18 feet of water, experienced usually when he had an upper respiratory infection and associated particularly with the late afternoon dive.

Eight weeks prior to consulting me, he had a prolonged upper respiratory infection, but continued to dive. Minor epistaxis after each dive was followed by intermittent bleeding from the left nostril for several days. While diving, he experienced severe pain in the left ear. On ascent from the tank, he experienced dizziness and noted a loss of hearing which had persisted in this ear, associated with a feeling of fullness, intermittent pain and tinnitus.

On examination, both tympanic membranes were slightly retracted, the reaction to the Rinné test was positive bilaterally, and the Weber test lateralized to the right ear. The tonsils were of moderate size, there was no excess of lymphoid tissue in the nasopharynx, the fossa of Rosenmueller was clear bilaterally, and a caloric test indicated normal vestibular function bilaterally. The audiogram showed:

	128	250	500	1000	2000	4000	8000
Right	20	10	15	25	20	25	15
Left	65	80	75	75	80	85	75

There had been no pre-employment audiogram. The patient had been a wartime instructor in the Air Force Tactical Training Command, but he denied any exposure to loud noises. On several subsequent examinations, there has been no change in the hearing. Both ears were inflated without difficulty, but the feeling of fullness in the left ear persists. I advised him to stop diving.

Comment. It is difficult to conceive of the degree of pressure change to which this patient was subjected causing a nerve deafness of the degree indicated by the audiogram. The lack of other evidence made it necessary for me to conclude that he sustained the deafness secondary to his diving activities. This case illustrates the value of pre-employment hearing tests in industry. Haines and Harris[21] emphasized the importance of these tests in evaluating the hearing following exposure to pressure. Anyone exposed to pressure either in employment or in recreation should have the hearing tested before and after indulging in such activity and every year thereafter.

Case 2. P. C., aged 26 years, a diver at the Marine Studios for seven years, complained in November, 1958, of fullness in the right ear since the ear became stopped up five months previously while he was diving. He experienced difficulty in clearing this ear, but continued to dive. Two weeks before I examined him, he had an upper respiratory infection and experienced great difficulty in clearing the ears while diving. On one dive to a depth of 12 feet for two minutes, severe pain in the right ear forced him to ascend and have a substitute diver take his place. He attempted to dive a week later and was unable to clear this ear. He had previously noted epistaxis, but had experienced only slight difficulty when diving with an upper respiratory infection. He admitted no exposure to excessive noises. Aware of the experience of the diver in *Case 1*, he was concerned about losing his hearing.

Except for a suggestive dullness of the right tympanic membrane, both tympanic membranes appeared to be normal. The reaction to the Rinné test was positive bilaterally, and the Weber test lateralized to the left ear. The nasopharynx was free of lymphoid tissue. Both eustachian tubes were readily inflated, but the right ear felt more stopped up after the inflation. The audiogram showed:

	128	250	500	1000	2000	4000	8000
Right	10	5	−5	0	5	30	0
Left	15	15	10	5	5	30	0

My diagnosis was subacute tubotympanitis, and he was advised to refrain from diving for several weeks. In January, 1959, he continued to complain of a tight feeling in the right ear. A week previously, on attempting another dive, he had experienced severe pain in this ear as he stepped off the ladder to the bottom of the pool. Since he could not equalize the pressure in the ear, he came to the surface and again experienced fullness in the right ear for several hours. Examination gave the same results as previously.

The use of radium in the nasopharynx was suggested to him, but declined when the inherent dangers and known effects of this treatment were explained to him. He elected to give up diving as a profession and perhaps act as a substitute diver at some future time should the difficulty clear up.

Comment. It is noteworthy that this diver experienced trouble after seven years of diving. He appeared to be emotionally stable and well satisfied with his work. There were no known compensation incentives.

A 24-year-old diver was observed immediately after ascending from a 40-minute dive to a depth of 12 to 18 feet at the Marine Studios. He had used a compressed air helmet, receiving compressed air at 25 pounds per square inch. His ears had blocked as he descended the ladder, but he was able to clear them by swallowing and noted no pain. Examination gave normal results. Examination of a diver, aged 23 years, resulted in normal findings under similar circumstances. Both men had been diving for approximately a year, had noted no hearing loss and had no difficulty in clearing the ears. Occasionally, however, they experienced pain in the ears and slight epistaxis when diving with a mild upper respiratory infection.

Aerosinusitis.

The etiology of aerosinusitis is similar to that of aero-otitis

media. The pressure differential between the sinus cavity and the ambient pressure is the deciding etiologic factor. Again, the severity of the sinus barotrauma is related to the degree and duration of the pressure differential. The larger sinuses are involved more frequently than the smaller ones. The maxillary and frontal sinuses are most commonly affected because of the small orifices. The length and tortuosity of the nasofrontal duct make the frontal sinus more susceptible to barotrauma. Any obstruction to a sinus ostium is conducive to aerosinusitis. On descent, the ostium is blocked and a differential in pressure ensues, resulting in a vacuum in the involved sinus. The negative pressure in the sinus must be equalized by some physiological mechanism. Engorgement of the blood vessels, edema of the mucosa, and hemorrhage in the sinuses result in equalizing the pressure in the sinus. When the space is filled sufficiently to equalize the pressure differential, the ostium will open. The symptoms may vary from a dull feeling in the affected sinus to severe pain. There may be hemorrhage from the nose in severe aerosinusitis.

Sound Transmission.

Sound is transmitted well underwater, traveling 4,800 feet per second in water as compared to 1,090 feet in air. It is, nevertheless, difficult to communicate with the voice underwater because of the difference in inertia and viscosity between air and water. Only about one part in 10,000 of the sound energy enters the water when one speaks into an ordinary mask.[19] For the scuba diver, speech is further complicated by the obstructing mouthpiece and the noise of escaping air during exhalation. Localization of sound is also more difficult under water than in air, probably because the high velocity of sound in water reduces by a factor of five the small difference in the arrival time of sound at the two sides of the head. The endolymph is largely mobilized through bone-conducted sounds.[37]

Air Embolism.

Air embolism is a potentially fatal accident of scuba diving. Lanphier[33] believed it second only to drowning as a cause of

death in this sport. In skin diving without a self-contained breathing apparatus, the air in the lung of the diver is compressed on descent as is the wall of the chest. On ascent, the pressure of the air in his lung returns to the ambient atmospheric pressure. This diver is not subject to an air embolism.

The scuba diver with his self-contained breathing apparatus takes into his lungs compressed air delivered at the ambient pressure of the water. If he ascends slowly, breathing regularly, the lung will equalize the pressure to the ambient pressure of ascent. If he holds his breath for one reason or another, such as ignorance, panic, or unconsciousness, the air in the lungs will expand with ascent, and if the ascent is from a great depth at a rapid rate, the alveoli are apt to rupture, and an air embolism is likely to develop. The expanding air on ascent increases the intrapulmonary air pressures sufficiently to overdistend the lungs and rupture the alveoli. The danger to the scuba diver is explained in Fig. 4.

There are two types of air embolism, pulmonary (venous) and systemic (arterial). A pulmonary air embolism results from a large amount of air entering the systemic circulation and going to the right side of the heart. Foaming blood containing large bubbles of air forms, which is more compressible than normal blood and less readily expelled. There is a retention of air following each systole. The presence of air in the right ventricle produces a loud churning sound, which is readily heard without a stethoscope, and is known as a mill wheel murmur. There is a resultant obstruction of the right ventricular outflow due to the air trap which forms in it. The pulmonary circulation is obstructed, and the right auricle and ventricle become distended. A pronounced rise in the venous pressure is followed by cyanosis and sudden death.

The position of the patient with a pulmonary air embolism may be life-saving.[16] Turning him on his left side permits the outflow of the right ventricle to be in the dependent position. The trapped air then disappears from the inferior position and is churned into a froth and mixed with blood in the right ventricular cavity. This may be a life-saving maneuver. Pulmonary air embolism may follow wounds or operation in which the dural sinuses are injured. The walls

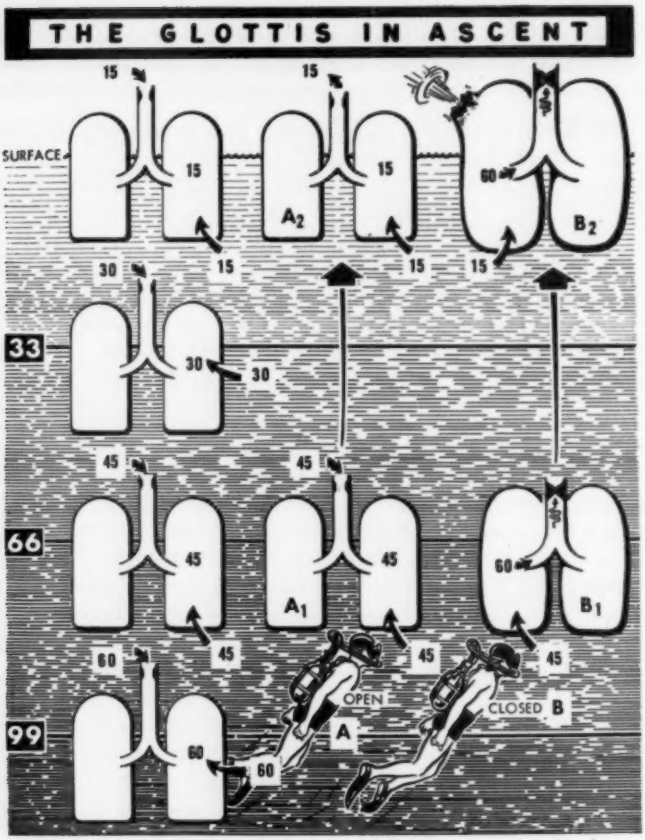

Fig. 4. The Glottis in Ascent. Divers descend with scuba to 99 feet (4 atmospheres of pressure). The lung remains the same. The lung pressure varies directly with the ambient pressure.

Diver A, ascending slowly, exhales with the glottis open. At the surface the lung pressure is equal to the atmospheric pressure.

Diver B panics because of defective equipment, closes the glottis to preserve air in the lung and makes a rapid ascent. With ascent the air in the lung expands. At the surface the atmospheric pressure is 15 pounds, and his lung pressure is 60 pounds. Rupture of the lung ensues causing air embolism, pneumothorax, or emphysema.

of these sinuses, being semirigid, favor the entrance of air. Replacing the fluid with air following an antral irrigation may result in a pulmonary (venous) embolism. Both situations occur in the field of otolaryngology.

Systemic (arterial) embolism results from air getting into the pulmonary venous channels. It is then propelled to the left ventricle and the systemic circulation. Ill effect is due to an embolic blockage of the cerebral and coronary vessels. This type occurs most frequently in scuba diving.

Kinsey[25] reported four cases of air embolism to illustrate the various symptoms and treatments. One man, after ascending 50 feet in the submarine escape training tank at the U. S. Naval Submarine Base in New London, was seen thrashing around on the surface, apparently in a convulsion. Frothy red fluid issued from his mouth. He was unconscious and in a convulsive state when taken to the recompression chamber nearby; shortly thereafter he died. Autopsy revealed widely dispersed air bubbles in all of the cortical blood vessels. Some vessels contained so much air that nearly all of the blood was forced out, leaving a 2 to 3 cm. expanse of blood vessel free of blood. Blebs filled with air were present over the pleura. Pronounced dilatation of the right auricle and atrium, and moderate dilatation of the left atrium and auricle was present, and there was air in the left auricle and atrium. In a similar case which came to autopsy after a fatal accident from air embolism, both the right and left auricles and ventricles were dilated by air. All major vessels encountered showed evidence of air, and the largest amount of air was expressed from the aorta and the superior vena cava.

Cases illustrating mediastinal and subcutaneous emphysema and mild cerebral vascular occlusion secondary to air embolism were cited by Kinsey.[28] In mediastinal and subcutaneous emphysema the alveoli are torn, and the air follows the pulmonary vessels to the mediastinum and then into the neck through the fascial planes. In cerebral embolism the air is carried to the left ventricle after entering small interstitial veins, producing arterial air emboli. The air causing dilatation on the left side of the heart is probably introduced through the pulmonary veins. In his case with air in both sides of

the heart, Kinsey[28] suggested that the air passed through the pulmonary lymphatic vessels to the large veins of the neck and thence to the right side of the heart. The large volume of air in the right auricle results in a cardiac tampon of blood (see Fig. 5).

Role of the Larynx. The larynx plays a particularly im-

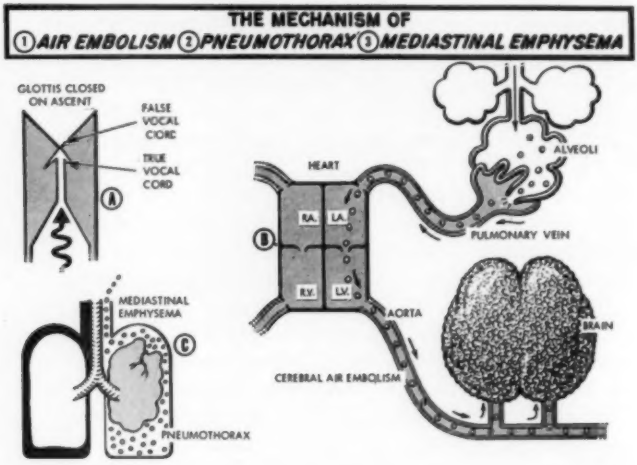

Fig. 5. The mechanism of (1) air embolism, (2) pneumothorax, and (3) mediastinal emphysema.

A. The glottis closes. The false cords prevent exhaling of air.

B. Increased lung pressure ruptures alveoli. Air enters the pulmonary circulation, passes to the left side of the heart and then to the cerebral or coronary circulation as an embolus.

C. A pleural bleb may form and rupture, forming a pneumothorax. Air may dissect along the pulmonary vessels or bronchi to the mediastinum to form a mediastinal and subcutaneous emphysema.

portant role in the etiology of air embolism. Phylogenetically, the larynx was most important in the evolution from water to land habitation. The most primitive larynx was found in the dipnoea, or lung fish.[46] It evolved because of the need for oxygen in these fish when existing in water with a limited or undependable supply of oxygen. Although the development of lungs capable of absorbing oxygen was a boon to them, it created the necessity for a means of keeping the water out

of the lung during submersion; otherwise, survival would not have been possible. In the lung fish there developed a simple circular group of muscle fibers at the upper end of the trachea to serve as a sphincter to close off the trachea during submersion. It is believed that this simple mechanism is the forerunner of the human larynx.

Phylogenetically then, it appears that the most primitive function of the larynx is one of protecting the lower portion of the respiratory tract, which is carried out by adduction of the laryngeal sphincter. It is ironic that the same mechanism which made possible the evolution of man from the sea may prove to be lethal to him during his return to the sea with his newly created breathing apparatus. The ingress and egress of air from the lungs are controlled by the valvular action of the true vocal cords and the false vocal cords. The true cords and false cords control the to-and-fro movement of air through the larynx. Because of the unique design, the valvular action is more powerful than the muscular sphincter effect. These functions are under both voluntary and involuntary control, supplied by the higher cortical centers and medulla in the brain. The true vocal cord acts as a valve which controls the ingress of air, and the false vocal cord controls the egress of air. The cortical centers allow one to hold his breath at will by adducting the cords until carbon dioxide is accumulated sufficiently to stimulate the respiratory centers of the medulla so strongly that the voluntary control is inhibited and the cords abduct as in forced expiration.[46] Reflex cessation of inspiration and tight adduction of the true vocal cords result when food or water or other foreign substances incite the sensory innervation of the larynx. False cords are tightly closed for the purpose of developing intrathoracic pressure by trapping air below the larynx. This mechanism is used in coughing, vomiting, defecation, urination, and parturition.

An emotional control is also exerted on the larynx. Beginning with the birth cry, the larynx reflects the emotional states throughout life. One's state of mind can frequently be diagnosed by listening to the voice. Fears and tensions are reflected by a high-pitched intense voice. The larynx

reverts to its protective reflex when provoked by one's physical and/or emotional condition. Stage fright and self consciousness are disastrous to the singer, and neurotic conditions are reflected in the neuromuscular reflexes of the larynx.[22]

While practicing with my scuba equipment in a swimming pool and learning to clear my mask, which consists of blowing water out of the face mask by positive pressure, I inhaled some water far posteriorly in my nose. This immediately set up a mass pharyngeal reflex and a mild laryngospasm, which persisted after I came to the surface. I talked in a high-pitched voice and had a paroxysm of coughing for several minutes. At a greater depth under less favorable circumstances than a swimming pool offered, such a sequence could be potentially fatal to as inexperienced a scuba diver as I was. It is essential for the scuba diver to learn to breathe through the scuba mouthpiece without his face mask.

Panic is the greatest hazard experienced by the skin and scuba diver. Several divers, in describing a condition of panic or near panic, have all volunteered a sensation of tension in the throat. Free ascent is a rapid ascent made when the scuba is defective, a diver exhausts his air supply, or his mouthpiece is dislodged. His cortical centers, if active, will abduct the cords so that air can be exhaled on his way to the surface. His emotional or reflex centers, stimulated by panic, may incite adduction of the cords because of his innate instinct to hold his breath on his way to the surface. If a laryngospasm due to inhalation of a small amount of water is superimposed, he will be a likely candidate for an air embolism. It is of the utmost importance that the diver be thoroughly familiar with the mechanism involved in the prevention of air embolism (see Fig. 4).

Treatment of air embolism consists of recompression in the recompression chamber under medical supervision. Recompression compresses the air bubbles to asymtomatic size, allowing the gas to return into solution and be removed by the blood. According to Lanphier,[33] failure to recognize air embolism or decompression sickness in a patient who has been using diving equipment is the most serious mistake possible.

The two may be indistinguishable, but both require recompression. Failure to recompress is apt to result in permanent injury or death. Physicians should know where the nearest recompression chamber is. Frequently it is many miles away. While the patient is en route to the chamber, he should be kept in a head low position to minimize the possibility of air entering the cerebral circulation. When air is in the right side of the heart, he should be placed in a left lateral position to displace the air trapped in the right ventricle.

Decompression Sickness.

Commonly known as bends and as caisson disease, decompression sickness is less of a danger to the scuba diver than is air embolism. The limited supply of air prevents his descending deep enough for a sufficient length of time to permit this illness to develop. It is unlikely to occur unless the diver carries several tanks or makes several deep dives in a single day. Gas dissolves in the body when under pressure and evolves when the pressure is released. Decompression sickness is due to the rapid release of nitrogen from the tissues on too rapid ascent. The gas evolves faster than the blood stream can carry it away and release it in the lung. Bubbles may form in the blood stream which may block the flow of the blood in the capillaries. Vail[50] reported deafness resulting from bubbles of nitrogen forming emboli or areas of necrosis in the internal ear. Permanent deafness may result. Shilling and Everley[50] presented evidence to show that even though decompression sickness may cause deafness, it rarely does so.

ANALYSIS OF SERIES.

The study of 38 divers here reported resulted from the interest stimulated by the earlier work of Taylor[51,53] pertaining to man's lack of adaptation to an aquatic environment. It has long been my desire to extend this research to the subaquatic environment. I first familiarized myself with the literature provided for the study of the skin and scuba diver. Then I received instructions in a swimming pool from two local physicians who were enthusiasts in both skin and scuba diving; also, I attended the meetings of a local club of experienced divers, known as the Jetty Jumpers.

In the course of studying a group of 38 skin and scuba divers, I submitted to them a questionnaire consisting of 39 questions and analyzed their replies. In addition, I performed a complete otolaryngological examination on all 36 men and two women in the group. The audiometric studies were carried out in a sound-proof hearing room (model 401, Industrial Acoustics) with a Beltone 15-A Audiometer. The divers, ranging in age from 16 to 44, were all experienced; 11 had been skin diving for over ten years and 18 for over four years. Observations were made on several of these divers at Troy Spring, a spring 100 feet in depth on the shores of the Suwannee river. The first observation was made after a descent to 33 feet and the next to 66 feet. The divers at the Marine Studios at Marineland on the east coast of Florida were examined before and after diving. My personal experience with barotrauma is included in the study. Observations made on the "mermaids" at Weeki Wachee Spring on the west coast of Florida are also reported, although these divers are not included in the series of 38 studied.

The enthusiasm of the skin and scuba divers in the series seemed to exceed by far that of the participants in other sports. This sport was more than an athletic contest to them. They were constantly seeking new knowledge; they were eager to learn about the physics of diving, physiology, marine biology, paleontology, archaeology and ichthyology. The Jetty Jumpers made it a practice to meet weekly throughout the year in their search for knowledge and to share experiences. They were fully aware of the dangers involved in this sport and made every effort to learn and stress the various safety factors.

It has been suggested that the relatively short, wide and straight eustachian tube of the child tends to make children more prone to ear infections. If this were true, one might assume that the person having the greatest number of ear infections in childhood would tend to have less difficulty in establishing patency of the eustachian tube when exposed to pressure. It is noteworthy that of the 38 divers, only six volunteered that they had any ear infections in childhood (see Table I). None of these experienced difficulty in auto-

inflation of the eustachian tube. They seemed, however, on the average, to have as much difficulty equalizing pressure in their ears while diving as the remainder of the group.

Barotrauma in Flying and Diving.

On the assumption that the individual responses to barotrauma in commercial flying and in swimming pools might be

TABLE I.

Past History of Otolaryngologic Difficulties.

	Yes	No
Frequent upper respiratory infections	11	27
Hay fever	8	30
Sinus trouble	12	26
Nasal obstruction, free of upper respiratory infection	8	30
Ear infections in childhood	6	32

TABLE II.

Observations Associated with Diving.

	Yes	No
Can descend to bottom of swimming pool without discomfort or pain in ears or sinuses	34	4
Difficulty equalizing pressure in ears while flying	4	34
Nasal bleeding during or following a dive	18	20
Perforated tympanic membrane during dive	3	35
Dizziness experienced during or following a dive	13	25
Tendency toward seasickness	10	28
Symptoms of decompression sickness following diving	1	37
History of panic	1	37
History of near panic	23	15
Encountered difficulty unable to handle without help while skin diving	7	31
Hearing loss following dive	9	29
Tinnitus following dive	16	22
Sinus involvement during or following a dive	20	18

of significance in their ability to withstand pressure, the divers were asked to give their responses to the changes experienced (see Table II). Of four who could not descend to the bottom of a 10 to 12 foot swimming pool without pain in the ears or sinuses, and four who had difficulty equalizing the pressure in their ears while flying, only one had difficulty in both flight and diving. With a slight upper respiratory infection they had more difficulty than those not having this

disability. The diver volunteering difficulty in both situations had to clear his ears every five feet down. Whenever he was unable to equalize the pressure adequately, he had intense pain in his ears. Always after diving, the left ear felt stopped up for about an hour and the right ear for approximately one week. His audiogram showed normal hearing.

One of the subjects having difficulty with flying had more difficulty with his ears than any of the other divers while I observed him after attempting a descent of 65 feet. He was unable to go deeper than 45 feet, and was one of the few I cautioned about the effect of skin diving on his ears. One of the eight divers was unable to autoinflate the eustachian tube and the other seven in this category had a little more difficulty than usual. They all had to clear their ears at less depth and more frequently than the divers not offering this complaint.

Teed[55] showed that observing the tympanic membrane while the subject autoinflates his ears is of significance in determining how a person will respond to pressure. I believe the person who has pain in his ears on descent in a pool and repeated difficulty in flying will show a poor response to pressure. A recompression chamber is not available to most physicians or divers to test their responses to pressure. I would tend to discourage the potential diver who gives such a history. The only true test, however, of response under pressure in a subaquatic environment is by diving. It is of extreme importance that divers be properly trained in autoinflating the eustachian tubes. The diver with repeated bouts of barotrauma should be discouraged from diving and subjected to depths only at which he is free of pain in the ear.

I believe that a person whose response to barotrauma both in flying and diving is similar to mine should be discouraged in this endeavor. I had anticipated my response while flying. I believe I shall be able to continue diving at shallow depths and also that at a much more gradual rate of descent, I might not have had the difficulty I encountered on the dive in which I attempted to reach a depth of 60 feet. Case 3 is a record of my personal experience.

Case 3. G. D. T., an otolaryngologist, aged 40, had a history of an initial and a secondary tonsillectomy and adenoidectomy at ages six and 13 years, and prominence of lymphoid tissue on the pharynx posteriorly and in the nasopharynx as a child. During his first two airplane flights, in 1941 and 1943, covering 1,000 miles with eight stops and made soon after having a mild upper respiratory infection, he experienced severe aural and sinus pain, followed by a bilateral serous otitis media, a loss of hearing of the conductive type and a bilateral low-pitched tinnitus of several days' duration. After a similar flight in 1945, serous otitis media persisted for several days and was worse in the right ear than in the left. Radium was applied to each side of the nasopharynx. During the next 12 years, various degrees of difficulty were associated with some ten or 12 flights varying in length from 150 to 1,500 miles.

In May, 1958, after a flight of 3,000 miles with four stops at a maximum height of 20,000 feet, he experienced severe sharp pain in the right ear and dull pain in the left ear on descent at the Los Angeles airport. Appreciable loss of hearing persisted for one week in the left ear and for two months in the right ear, and there was a low-pitched roaring tinnitus. During sleep the right ear would clear, but when he was operating the following morning with his head in the dependent position, the feeling of fullness would recur as the fluid entered the eustachian tube, and it would persist all day. For removal of fluid from this ear, paracentesis with inflation was resorted to during the fifth week and repeated ten days later.

After three months had elapsed, this experience led to testing the response in a subaquatic environment. Diving in a pool to a depth of eight or ten feet resulted in a feeling of stuffiness in the ears and a roaring tinnitus lasting several hours. Early in November, at Troy Spring, on descending gradually to 40 feet with scuba equipment, he experienced little difficulty in inflating the left eustachian tube but much difficulty inflating the right tube. On ascent, pain suffered at the 20 foot level led him to descend five feet before reascending. At this point, the egress of air through the tube was distinctly audible. Both tympanic membranes were inflamed, and tinnitus persisted bilaterally for 48 hours.

In mid-November, on making a more rapid descent at Troy Spring than previously in an attempt to reach the 66 foot level, he experienced pain at the ten foot level, could not open the right tube at the 40 foot level, and at the 45 foot level suffered pain so severe that he made a gradual ascent. On-the-spot tests were made with the AMBCO Otometer before and after diving, and the hearing was above the 5 db level for each frequency both before and after the dive. The left tympanic membrane was injected after the dive and the right one inflamed. The next day the right ear felt as though it were full of fluid. Two days later the left ear was open, but the right ear felt tightly closed. Bilaterally, a roaring tinnitus persisted. The left tympanic membrane appeared normal, and the right tympanic membrane showed evidence of hemorrhage. The ear was filled with fluid at that time and also two days later when the evidence of hemorrhage had subsided. Ten days later the fluid had disappeared, and the tympanic membrane had returned to normal. Immediately following this dive, mild bleeding also occurred from each side of the nose and continued for several minutes after he came to the surface. The first nosebleed he had ever experienced probably was caused mostly by pressure change in the sinuses and nose and the pressure of the face mask against the nose during frequent attempts to clear the ears.

Early in December, at Marine Studios, he descended in a tank to a depth of 12 feet, wearing a diver's helmet and a rubberized waterproof suit with 25 pounds of pressure fed through the compressed air hose.

There was no appreciable change in the ears on descent or while down, but on ascent the eustachian tubes were blocked and remained so for two days. The right ear felt blocked for a week, but there was no evidence of fluid in the ear.

At Mayport, Fla., in mid-December, compression to a pressure equivalent of eight feet of water, in a recompression chamber, resulted in a completely blocked right tube at ten feet produced pain. On flying, he had always noted that at an altitude above 7,000 feet he could tell when the plane started down, and he experienced gradual loss of hearing as the plane descended. Despite yawning, swallowing and autoinflation, he was never able to open the eustachian tubes adequately. He had also used antihistamines and nasal decongestants, but to no avail.

The audiograms, made periodically, showed:

May 30, 1958—

	128	250	500	1000	2000	4000	8000
Right	20	15	10	5	5	10	15
Left	5	0	5	5	–5	0	10

June 23, 1958—

	128	250	500	1000	2000	4000	8000
Right	20	10	5	5	15	20	35
Left							

October 6, 1958—

	128	250	500	1000	2000	4000	8000
Right	10	5	0	–10	0	10	10
Left	15	10	5	10	0	0	10

December 8, 1958—

	128	250	500	1000	2000	4000	8000
Right	20	15	5	0	0	30	35
Left	10	0	0	5	0	0	5

December 9, 1958—

	128	250	500	1000	2000	4000	8000
Right	20	15	0	0	0	20	20
Left	10	0	0	0	0	0	5

January 12, 1959—

	128	250	500	1000	2000	4000	8000
Right	5	–10	–5	–5	–5	10	5
Left	5	–10	0	-5	–10	0	5

It is obvious from this record that his experiences with severe barotrauma during the preceding seven months resulted in no permanent damage to his hearing.

Nasal Bleeding.

Eighteen of the 38 divers examined in this study had ex-

perienced nasal bleeding during or following diving. In the one severe instance, the diver had an upper respiratory infection, and the source of the bleeding was believed to be the frontal sinus. This bleeding may result from the forced pressure of the face mask against the nose in an attempt to inflate the eustachian tube. It may also result from a negative pressure in the nose created by improperly inflating the mask on descent by positive pressure from the nose to equalize the external pressure. In none of those having frequent nosebleed was there noted a prominent vessel on the septum which might have caused the bleeding.

Perforated Tympanic Membrane.

Three divers presented a history of a known perforated tympanic membrane associated with diving. In all, the lesion healed without treatment. In making a rapid descent, if the diver is unable to equalize pressure in the middle ear with the ambient pressure, the eustachian tube will block completely. If he continues to dive after experiencing the sensation of pain, the tympanic membrane may suddenly rupture because of the increased external pressure. Rupture is usually accompanied by pain which is not necessarily great. There is the sensation of a change in tension, and a noise is heard in the head. Severe dizziness may ensue from the introduction of water into the ear, setting up a caloric response in the semicircular canals.

Case 4. J. C., a man 34 years of age with ten years' experience in skin diving, had had limited experience in scuba diving. In 1946, while he was skin diving and spear fishing, he descended suddenly after noting a gar fish. At a depth of eight to ten feet he experienced pain in the ears, but continued to descend without clearing them. Sudden sharp pain in each ear ensued. He then ascended and felt as though he were flopping around from side to side, more of a to-and-fro sensation than a true vertigo. Both ears bled for a short time, and the pain persisted for two or three hours, but there was no appreciable loss of hearing. At the Navy Dispensary he was told that both tympanic membranes were perforated. He stayed out of the water for two or three weeks, and the perforations healed without treatment. He believed he could have reached the surface without difficulty even from a much greater depth. In 1948, the left tympanic membrane was perforated by shock from the firing of a large gun on shipboard. He was able to blow smoke through the perforation, but experienced pain each time. There was a loss of hearing for several days following this perforation, which healed without treatment.

In December, 1949, while at the Submarine School in New London, he made a free ascent from 100 feet and then dived down into the tank

to a depth of ten to 15 feet, with perforation of the right tympanic membrane resulting. The lesion, which was preceded and followed by pain, was confirmed by medical examination and healed without treatment. In 1951, an attempt to dive to a depth of 17 feet resulted in perforation of both tympanic membranes. He noticed that in each episode the pain was worse after the tympanic membrane ruptured. While in the Bahamas diving for lobster in 1958, he experienced pain at a depth of 18 feet, but he went "the extra foot" without clearing his ears, and the tympanic membrane was again perforated. He got the lobster after perforation of the tympanic membrane.

In November, 1958, an audiogram showed:

	128	250	500	1000	2000	4000	8000
Right	10	5	0	5	5	55	15
Left	5	5	5	10	0	50	20

Examination of the ears revealed atrophic areas involving most of the posterosuperior quadrant of each tympanic membrane. With autoinflation there was a protrusion outward of these atrophic areas.

Comment. It is probable that the outward protrusions were the site of the perforations in this case. The dip at the 4000 frequency in each ear was probably due to trauma associated with the exposure to gunfire during the patient's eight years of service in the United States Navy. Behnke[5] noted a few proved cases of deafness arising from pressure trauma in contrast to the permanent aural damage caused by the exposure to the auditory trauma of gunfire.

Case 5. C. M., aged 30, experienced in skin diving for 20 years and in scuba diving for seven years, had three episodes of a perforated tympanic membrane secondary to diving. The first bout occurred in the right ear at a depth of 40 feet, when he failed to clear the eustachian tube properly. Pain and severe vertigo followed, the vertigo lasting for several minutes after he was out of the water. A physician irrigated the ear with a solution which he believed contained mercury and also inserted grain alcohol drops into the ear. He experienced severe vertigo during the irrigation and severe pain with the insertion of the alcohol. The ear became badly infected and was washed out almost daily, the irrigation causing vertigo and pain each time. The drainage continued for several weeks. The physician informed him the perforation was still there and would always be present, and advised him against ever getting the ear wet. Another physician, a year later, could see no evidence of perforation. He started swimming again with no ill effects.

Perforation of the left tympanic membrane occurred again the next year when he was at a depth of 25 feet because he failed to clear the ears properly. He experienced immediate pain and vertigo. Under treatment with sulfonamides and penicillin the ear healed in about two weeks. Several months later, perforation of the right tympanic membrane with associated vertigo and pain occurred again while he was diving. The vertigo lasted for about one minute after he got out of the water. He was treated with antibiotics by another physician, who cauterized the

ear after five days and again ten days later. Healing was accomplished in three or four weeks.

He then remained out of the water and refrained from flying for about two months. He stated that a hearing test showed excellent hearing in both ears at both ends of the frequency range several months after the last perforation. His physician told him that his hearing was much better than that of most of the people he tested. In my study of this diver no audiometric test was made. Examination of his left ear, however, revealed a healed posteroinferior perforation with an area of atrophic membrane at the site of the perforation. The right tympanic membrane showed a large posteroinferior healed perforation with an area of atrophic membrane at the site.

In every instance of perforated tympanic membrane in this series there was a history of vertigo following the rupture (see Fig. 6). Haines and Harris[21] observed that a rupture of the tympanic membrane produces a loss of hearing not greater than 5 or 10 db.

Case 6. J. T., a man aged 32 years, one of the most experienced divers questioned, was experimenting in 1950 with ear plugs in each ear while scuba diving. At a depth of 20 feet he experienced severe pain in the left ear, followed by a loud roaring sound. When he surfaced, the left ear bled for several minutes. Upon removal of the ear plug, he suffered severe pain when aerating the ear. He consulted an otologist at once, who told him the posteroinferior portion of the tympanic membrane was perforated. There was little change in the hearing after the perforation. The rupture probably resulted from a differential pressure developing in the air space between the tympanic membrane and the ear plug. The increasing pressure in the middle ear due to air from the scuba equipment under pressure probably perforated the tympanic membrane from within.

Examination of his ears in November, 1958, revealed no abnormalities. There was no evidence of scarring or atrophic areas on the tympanic membranes.

An audiogram showed:

	128	250	500	1000	2000	4000	8000
Right	5	0	−10	0	0	60	45
Left	5	5	−5	0	0	55	60

The high frequency loss shown in this audiogram was probably due to exposure to acoustic trauma sustained while he was in the armed services.

One should never wear ear plugs while skin or scuba diving. With a tight-fitting ear plug in the external auditory canal, a vacuum will develop between the plug and the tympanic membrane resulting in a differential of pressure. If the pressure of the scuba delivering air into the mouthpiece is equal to the ambient pressure of the water which is pressing on

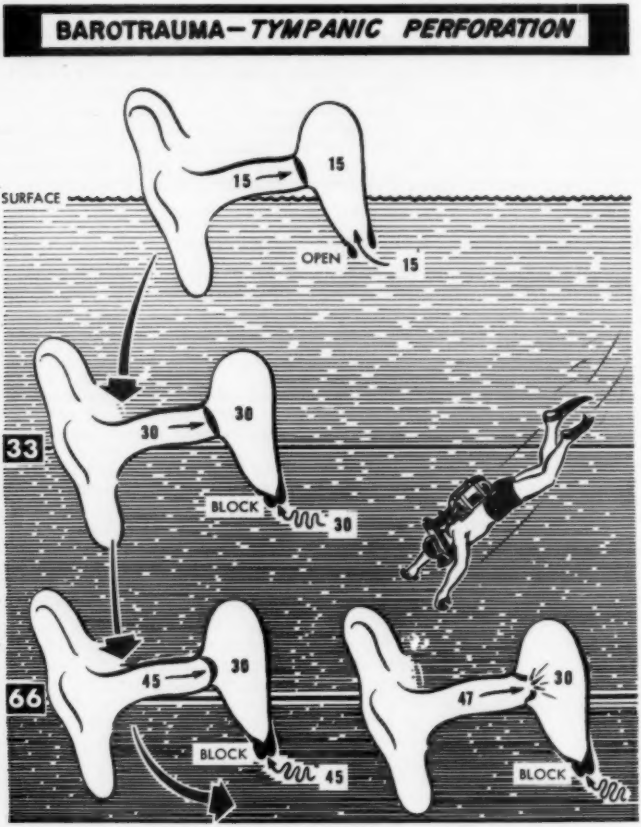

Fig. 6. Barotrauma—Tympanic Perforation.

A. At the surface the eustachian tube is open, and the pressures are equalized.

B. At 33 feet the pressure is equalized. Blocking of the eustachian tube begins. Pain ensues.

C. At 66 feet the diver ignores the pain and continues the descent. The pressure is not equalized. The tympanic membrane is pressed inward.

D. The tympanic membrane ruptures inward because of the difference of middle ear and ambient pressure.

the external surface of the plug, the tympanic membrane may rupture outward as the pressure in the middle ear increases. If there is a blockage of the eustachian tube and the pressure is less than the ambient pressure, the plug may be pushed inward through the tympanic membrane in skin diving and in scuba diving (see Fig. 7).

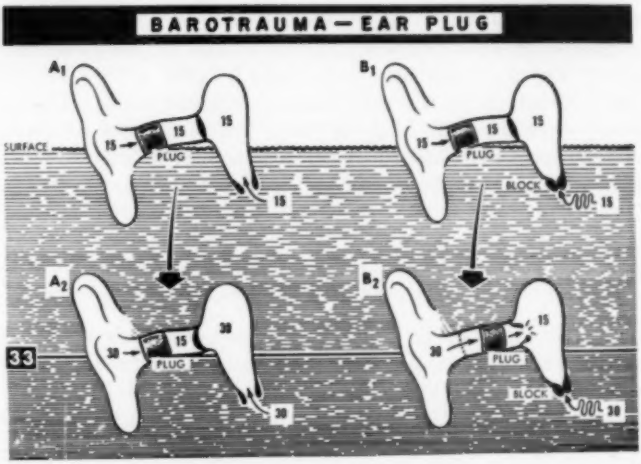

Fig. 7. Barotrauma—Ear Plugs.

A-1. Ear plug in the external auditory canal; pressure equalized. Wide open eustachian tube.

A-2. Pressure differential in the canal between the ear plug and the tympanic membrane. As the middle ear pressure increases, the tympanic membrane bulges outward. This bulging will lead to perforation if the pressure differential increases.

B-1. Ear plug is in the canal; the pressure is equalized. The eustachian tube is narrow.

B-2. The eustachian tube is blocked. The ambient pressure forces the ear plug deep into the canal, and the pressure ruptures the tympanic membrane.

Dizziness, Seasickness, Bends, and Panic.

Thirteen of the 38 divers had noticed a variable degree of dizziness associated with diving. One stated that if he ascends faster than at the rate of 25 feet per minute, he becomes dizzy. Ten were prone to seasickness. The three who experienced motion sickness along the bottom of the sea

attributed it to positional change due to the movement of the water while their eyes were fixed on the bottom as they searched for objects. One highly experienced diver who had dived to a depth of 200 feet acknowledged symptoms of decompression sickness. A most experienced and seemingly stable diver admitted having experienced panic on three occasions, once when left by his companion 400 feet back in an underwater cave, again when he lost his sense of direction in a spring, and a third time when he lost his visibility in a spring on another occasion. Twenty-three of the 38 admitted situations in which they approached panic while 15 stated they had never been near panic in any situation. The incidence of panic is probably greater than this study indicates, as divers are doubtless reticent about acknowledging it. Seven divers had encountered difficulties while diving that they were unable to handle without help. Of these, five admitted near panic.

Loss of Hearing Associated with Diving.

The nine divers in the series who noted loss of hearing during and following diving, attributed it to inability to equalize pressure through the eustachian tube. The loss lasted from several hours to several days and varied in degree proportionately with the rapidity, duration and depth of descent. None believed that they had sustained any permanent loss as a result of diving.

Audiometric studies were carried out on the 38 subjects. In two, a diagnosis of otosclerosis was made; they were the only ones in the series who were conscious of any permanent loss of hearing. A composite audiogram showed high frequency losses, but most of these were, in my opinion, due to various exposures to acoustic trauma. Dr. Kenneth Wilson,[60] Director and Audiologist of the Speech Correction Clinic of Jacksonville, evaluated the 38 audiograms and made the following subjective observations:

1. The group generally has hearing acuity within the limits of normal variation.

2. A differential effect on hearing acuity in specific cases

is noted. These cases can be attributed to pathological (or basic physiological) conditions, such as noise trauma, etc.

3. Possible implications: Some of these skin divers represent cases of hearing impairment not directly related to diving. The physician should be alert to the need for advice concerning the possible effects of continued skin diving on the hearing impairment.

Nine divers descended to a depth of 33 feet (two atmospheres) at Troy Spring for a duration of 20 minutes. Hearing tests, made with the AMBCO Otometer before and after the dives, indicated no appreciable change in hearing in any diver. In each diver the tympanic membrane was injected; also, the vessels in the region of the malleus and the posterosuperior portion of the tympanic membrane were injected, especially the vessels near the annulus. In two of the divers there was a fullness posterosuperiorly. The tympanic membrane was protruding in this area due to the increased pressure following inflation of the eustachian tube during the dive.

Three weeks after the descent to 33 feet, ten divers were observed before and after descending to a depth of 66 feet for 15 minutes. The blood vessels on the tympanic membrane were injected in the same area as after the 33 foot dive. Except in one instance there was no definite change in hearing. In one diver there was a loss of 15 db at the 4000 cycle in each ear, and at the 6000 cycle there was a 30 db loss in the left ear and a 10 db loss in the right ear. The tympanic membranes were markedly inflamed, and there was fullness posterosuperiorly. He was able to descend only to 40 feet because of severe pain and great difficulty in clearing his ears. Ten days later his hearing had returned to his normal level. Injection of the malleolar vessels on each tympanic membrane was still present.

Tinnitus.

Sixteen of the divers had noted a ringing sensation in their ears. Of these, six did not relate the tinnitus to diving, and four had been subjected to sufficient acoustic trauma to give them the persistent tinnitus. One proficient diver, aged 30, had had a persistent roaring tinnitus since 1957.

Sinus Involvement.

Twenty of the divers had sinus pain either when diving with a cold or when descending too rapidly, but it did not seem to be of too great significance to any of them. One had severe bleeding for a few minutes following a dive.

Appearance of Tympanic Membrane.

Prominent dilated blood vessels were observed along the malleus in seven divers. In three, there were also vessels posterosuperiorly at the annulus. These divers were examined in the office and none had been diving for at least a week. This finding probably represents a chronic state of congestion in these vessels, which might result from the pressure differential developing with diving or the frequency of autoinflation. The congestion seemed to be more pronounced in the ear which was the more difficult to clear. I do not recall observing this condition in any other type of patient. These vessels are the ones which are seen to be injected following diving.

TABLE III.

Greatest Diving Hazards in Opinion of Divers.

Panic	23
Ignorance	4
Foolhardiness	3
Other careless divers	3
Poor training	2
Air embolism	2
Bends	1

The posterosuperior portion of the tympanic membrane seemed most involved. After a scuba dive, a ballooning out of this area, due to the increased pressure in the middle ear, was seen in some divers. This area appears to bear most of the brunt in tubal inflation. A flaccid atrophic area was present posterosuperiorly in four divers. This area, well demonstrated on inflation, is the area which is seen protruding outward after inflation, following diving. Campbell,[56] in discussing Teed's paper, stated that frequent autoinflation or politzerization does not lead to flaccid tympanic membranes.

Miscellaneous.

Eighteen divers had more difficulty clearing their ears when free diving, and nine when scuba diving; 11 noticed no difference. There were 20 smokers and 18 nonsmokers in the group. In regard to the relationship of diving to respiratory infections, one noticed an increase, eight a decrease and 29 no change.

Divers' Observations.

In the opinion of the divers, the greatest hazards to life resulting from diving are man-made; namely, panic, ignorance, foolhardiness, other careless divers, poor training, air embolism and bends (see Table III). Thirteen had learned skin and scuba diving by self instruction through reading books and then diving, 21 by diving with experienced divers and only four by organized supervision. To prevent fatalities secondary to this sport, it would seem most important that organized supervision be encouraged.

One diver admitted that in most every dive "something will surprise you and scare you half to death." One of his worst scares was when a six-inch fish swam into his mask while he was working around some piling. Despite the frightening experiences, he acknowledged no inclination to panic and related that he tested himself to see how he would respond to panic situations. He had gone down in 30 feet of water and then gone into the mud to a depth of 10 feet as a proof of stability. Believing that cave diving gives one confidence, he had gone far back into a cave tunnel, shut off his source of light, taken off his mask and removed his mouthpiece. He had further tested himself by ascending from great depths with his mouthpiece out.

Several divers commented that the more they dived the easier it was to clear their ears; if they skipped two or three weeks, they had greater difficulty, and at the beginning of the season they always experienced the greatest difficulty. One diver observed that when he was skin diving at five feet or deeper and swimming parallel to the bottom of the ocean with one ear higher than the other, he would always experience greater pain in the dependent ear. Many of the

divers volunteered the comment that it is most important to clear the ears gradually and thoroughly during the first ten to 20 feet of descent. One thought that once he had passed 33 feet (two atmospheres absolute) and had cleared his ears properly, it mattered little how deep he went as far as the pressure in the ears was concerned. Gradual descent is important so that the air may flow through the eustachian tube to equalize the pressure in the middle ear. According to Boyle's law, the volume of gas which must pass through the tube is less for the same depth change at greater depths below the surface. The diver, therefore, does not have to stop his descent so frequently to equalize pressures below the depth of the first pause at ten or 15 feet. When the pressure is equalized in a well ventilated tube, he probably will not have to stop again for 40 to 60 feet, and then not again unless making a dive beyond 120 feet if the pressures were completely equalized at each pause.[37] One diver mentioned that dark or black water made his throat "tighten up," and another said that when he was frightened or emotionally upset, his throat felt closed. A woman diver was of the opinion that women are just as good at skin and scuba diving as men.

OBSERVATIONS AT WEEKI WACHEE SPRING.

Displaying courage, grace and beauty in a magnificent underwater show, the "mermaids" at Weeki Wachee Spring demonstrate admirably how well one may adapt to an aquatic environment with proper precautions and training. Their underwater acts are so well carried out that it is difficult to believe they are submerged. Trained by Mrs. Charles R. McNabb, herself an excellent performer, they know diving physics as well as diving physiology, and are fully aware of the effects of pressure on the human body. They are not allowed to dive with any sign of an infection of the upper respiratory tract and must surface at once and be replaced if they are unable to clear their ears or sinuses during a performance. Usually, they present one or two shows a day, but sometimes as many as four. Their average age is 19 years, and they come from all parts of the country.[39]

The show consists of various acts taking place underwater

continuously for 30 minutes. The performers present an excellent display of control of buoyancy by the amount of air inhaled and exhaled; as they breathe out, they descend, and as they inhale, they ascend, with no movement of the hands or legs. They breathe through a hose which delivers air pressure at 88 to 120 pounds per square inch. By proper control of breathing and swallowing, they eat a banana underwater with ease and then drink a bottle of soda pop. The graceful ballet they perform illustrates excellent breathing control. One diver makes a deep dive and then prolongs ascent without the air hose by engaging in ballet antics as she constantly exhales on the way to the surface.

The performers have less respiratory infection than employees not diving. The greatest difficulty is with the ears and sinuses. Alcohol drops are introduced into the ears after every dive. It is estimated that these girls are unable to dive 10 per cent of the time because of upper respiratory infections or other illnesses.[39]

At the time my studies were made, the air temperature varied from 61° to 70° F., and the water temperature was 74.2° F. The most striking observation was the great loss of body heat experienced by each diver. The temperature changes in four subjects before and after they had been completely submerged in water for 30 minutes were:

	Before	After
F. M. C.	99.2	93.2
Q. A. S.	99.6	95.0
N. N.	98.0	94.8
M. W.	99.0	93.4

There was an average loss of 4.85 degrees. The temperature was taken just before descent and immediately on coming out of the water. That these girls were all very cold was manifested by shivering, chattering teeth, goose pimples, and a reddish-purplish hue to their skin. After being exposed to such temperature change one to four times daily five or six days a week, it is surprising that they have a low incidence

of infection. They take a hot shower shortly after coming out of the water and then dress in warm quarters.

Noting that the human organism must maintain a constant average temperature of 98.6° F., Taylor and Dyrenforth[54] concluded that any considerable degree of deviation from this average for appreciable periods of time will result in morbid changes in the body. They further demonstrated that cold water has a veritable appetite for heat. Since the ratio of conductivity of water to air is 27 to 1, it follows that water takes heat from the body 27 times faster than does air. Almost all heat transfer under water is dependent upon conduction of heat from the skin to the water. The body loses more heat than it produces. The degree of heat loss under water is determined by the temperature of the surrounding water, the resistance of the body surfaces, and the resistance of the material worn. Calories must be consumed to help in maintaining a constant temperature.[37] The colder the water the greater the caloric consumption. This increases the metabolic rate, increases the need for air consumption in scuba diving, and leads to fatigue and exhaustion.

Man lacks a compensating mechanism for the maintenance of an average normal temperature such as is seen in aquatic birds and animals. These girls wear regular bathing suits. Their only protection is not to stay underwater too long and to restore their normal body temperature as soon after they dive as possible. Man's ingenuity has been an aid in allowing him to compensate somewhat for his temperature changes in water. Two types of suits have been designed, the wet suit and the dry suit. The wet suit is constructed of rubber or foam neoprene material and allows a small amount of water to enter the suit, which is not able to circulate. The body heat warms this small amount of water, and it acts as an insulating barrier against the cold water. Air affords better insulation than water. The dry suit is an air-tight, tight-fitting rubber suit under which woolen underwear can be worn. The air in the suit is warmed and helps maintain the body heat. Should these girls abandon their present mode of attire with its aesthetic appeal and wear these suits, they would be better able to maintain their normal body tempera-

ture, but would find it more difficult to attract the present audiences.

The ears of seven girls were examined before and after diving. Several of them made two or three dives during the day. Hearing tests were made under poor testing conditions, with a Beltone 10-A Audiometer. There was no appreciable change noted before and after diving. In all but one, the tympanic membrane showed no significant change. There was a mild injection of the blood vessels posterosuperiorly and along the malleus, as was observed in the series of 38 divers studied. In the girls going to the greatest depths more injection of the vessels was present than in those participating in the more shallow dives.

Case 7. N. N., a 23-year-old diver who has been skin diving for six months, stated she had frequent colds and believed "sinus trouble" had developed since she began to dive. Four months previously she experienced pain in the right ear, followed by a feeling of fullness and a loss of hearing, evidently an aero-otitis media with formation of fluid in the middle ear. The condition necessitated a paracentesis by a physician on four different occasions during a period of one month. Since then, on diving, she had had a great deal of difficulty in clearing the right ear, especially during the first ten to 20 feet of descent, and had experienced an occasional pain in this ear, especially if she had any sign of an upper respiratory infection. It is noteworthy that she had less difficulty clearing her ears with her head up than when in the dependent position.

She complained of frequent pressure over the eyes and pain in the head and nose. Otoscopic examination of the right ear before diving revealed an injection of the malleolar vessels. A prominent vessel was present on the anterior edge of the malleus and one on the posterior edge. A dilated vessel was prominent posterosuperiorly at the annulus. There was slight injection of the malleolar vessels in the left tympanic membrane. After the first dive there was no change in the left ear. In the right tympanic membrane there was moderate retraction, and the vessels were markedly injected over the malleus posterosuperiorly. I observed this ear at one hour intervals for the next six hours. She made one other dive during this period. The right ear was improved after the second dive. Several hours later the ears had the same appearance as they had before the dives.

Comment. This diver represents the type of person I think should give up diving. I believe that if she continues to have trouble equalizing pressure in her right ear, she will be apt to suffer a hearing loss. At the present time she has no hearing impairment. Comparison of the findings on her ear after the first and second dives appeared to indicate a tube in which the pressure had not been equalized.

Dr. Maurice Haddad, who has observed the girls at Weeki Wachee Spring for six years, reported external otitis as the most common complaint. During the Summer, when it occurs most frequently, he sees about one girl a week, but the use of ear drops, before and after swimming, has reduced the incidence of this disease. He encounters aero-otitis media only occasionally, and rarely in acute form; he estimated that he performs myringotomy six or eight times a year in treatment. Frontal sinusitis with a vacuum type of headache is not uncommon. Three or four of the girls have a high septal deflection. He performed a submucous resection on two of them this year, which gave relief. In his opinion, they have an inflammation of the eustachian tube, following which they experience vertigo underwater. Two of them lost their sense of direction, and one tended to have a fainting spell. He attributed these reactions to a pressure phenomenon in the middle ear, but did not believe there was subsequent loss of hearing. Most of the girls stay on the staff for only one or two years. He concluded that they have no more difficulty than the general population except for respiratory infections. He performed an adenoidectomy during the past year on two of them.

CONTRAINDICATIONS.

Difficulty in equalizing pressure in the ears and sinuses is the main contraindication to skin diving and scuba diving from the otolaryngological standpoint. Inability to inflate the eustachian tube properly is probably the greatest limiting factor to this form of diving. A person who has this difficulty on going to the bottom of a pool or while flying will probably have difficulty with diving. A perforated tympanic membrane is a definite contraindication. The danger of vertiginous attacks secondary to water getting into the middle ear could be a serious consequence. A person with dizziness secondary to endolymphatic hydrops or other etiological factors should not dive, and one with chronic recurrent external otitis would have a difficult time. Unless a person is in good general health and capable of standing up under emotional strain and physical duress, he should not indulge in this sport.

DISCUSSION.

Carefully supervised instruction in skin and scuba diving by experienced divers is most important in the prevention of accidents and death from this sport. Rigid tests should be passed by the student before he should consider himself capable of this potentially dangerous activity. The potential dangers of scuba diving have been taken entirely too lightly. Television, motion pictures and magazines have brought the fascination of this sport to millions of enthusiasts, but too little of the potential dangers have been made known to them. Local skin diving clubs should set up carefully controlled and rigid courses for skin divers, and certification should be the aim of these local and national skin diving associations and such organizations as the American Red Cross. The Conference for National Co-operation in Aquatics has made available a book[48] entitled "The Science of Skin and Scuba Diving," which serves as an excellent training manual and source of information for all divers. The increasing popularity of this sport indicates that some form of certification must be set up if potential deaths are to be prevented.

The U. S. Divers Company,[47] makers of the Aqua-Lung, find the heaviest participation in scuba diving in California, Florida, New England, the Greater New York and Upper New York State area, Michigan, Oregon and Washington, in the order named. This company would not attempt to give an accurate figure on the number of skin and scuba divers in the United States at the present time, but estimated there are several million. Its sales volume has doubled in the past year.

The United States Public Health Service has no statistics on the number of deaths occurring annually in this country due to skin and scuba diving. It would be of interest and important to have this annual death rate. In 1956, 6,139 persons drowned in the United States. Lanphier[31] stated that there is no civilian information on any reliable scale pertaining to the mortality figures of the skin divers. It is his impression that the actual rate is much lower than the extent and average quality of sports diving activity would lead him as

a submarine medical officer to expect. The death rate in Navy diving is probably less than one per year. As participation in the sport of skin and scuba diving continues to grow, the death rate will be kept down only if there is adequate training for prospective divers.

The difference between the upper part of the respiratory tract of man and that of the aquatic mammal lies not in the respiratory membrane, but in the aquatic mammal's ability to close the nares voluntarily or involuntarily. Taylor[53] demonstrated that the aquatic mammal's most persistent modification for life in the water is the ability to close the external nares and protect the respiratory epithelium from the destructive action of water. Man's rudimentary muscles for this purpose are now of value only in facial expression. He then showed that man's one natural means of protecting the nasal mucous membrane while swimming is by the proper method of breathing. By exhaling through the nose while the head is submerged and inhaling through the nose while the head is above water, the swimmer tends to maintain a proper air pressure in the nasal cavities, thereby protecting the sinuses and eustachian tube from water.

Such a mechanism is more important in a subaquatic environment. The face mask allows man to protect the nasal mucous membrane from the deleterious effects of water by inhaling and exhaling through the mouth without inhaling through the nose, which is sealed air-tight from the water by the pressure of the mask. The mask also allows for excellent vision under water, which is not possible with the cornea exposed to the water. The skin and scuba diver should know the proper method of using the mask and should be familiar with clearing the mask under water. He must be prepared for the eventuality of his mask being accidentally displaced or broken. Under such circumstances he must be able to breathe through his mouth with his nose not covered or occluded. He does so by elevating the palate and occluding the posterior nares. He also keeps a positive pressure in the nasal cavities by inhaling from time to time through his nose. He should be able to pick up the mask underwater, put it on

his face and blow the water out of it. This maneuver could be life-saving on occasion.

One need only to read of the exploits of the Navy underwater demolition teams to realize how well man has been able to adapt himself to an aquatic and subaquatic environment. Yarbro[61] believed that the main problem of these underwater swimmers today is countermeasures initiated and imposed by the enemy. In describing the tremendous handicaps and physical strain they must endure, he concluded that this military vocation requires a stout physique, a stout intellect, and a stout heart.

One of Florida's most experienced skin divers, Charles R. McNabb,[38] recently related his experience in removing the bodies of two 17-year-old divers from Salt Spring in western Florida, near Weeki Wachee Spring. Three divers entered a narrow undeground stream 125 feet deep, wearing standard tanks with enough air for only a very short time at this depth, and carrying no guide line to lead them back to the surface. Describing the tragedy in which two of them met death, he said: "I can only hope that God grant every diver a mental picture of the two bodies as I saw them lifeless in their watery tomb. Know your equipment and know your own limitations. Life is too precious to waste . . . There are far too many people today that, having purchased diving equipment and learning to breathe from it, decide they are divers. Those of us that have been diving all these years, both for sport and as a profession, know the hazards and have been properly trained in the use of the equipment. Until one has mastered the emergency techniques, such as clearing your mask, clearing hose, breathing from another diver's mouthpiece, ditching equipment and making a free ascent, etc., *you are not a diver*, but a potential accident. But most important of all after you have learned to use the equipment properly, *use good judgment.*"

SUMMARY AND CONCLUSIONS.

If man will understand and accept the limitations nature has placed on him in an aquatic environment, he may learn to adapt himself well to skin and scuba diving. He need be

limited only by the physiology and physics which govern his being, and the amount of air his equipment makes available.

Man has few structural adaptations for an aquatic or subaquatic environment. Proper methods of breathing and intelligent use of the face mask, fins, snorkel and scuba allow him to adapt to the aquatic world.

Loss of hearing resulting from aero-otitis associated with increased pressure is not common. When present, it is the result of damage to the middle ear caused by the inability to equalize air pressures. The person having difficulty equalizing pressure through the eustachian tube should not indulge in these sports.

Skin diving and scuba diving are potentially fatal sports. All participants should be thoroughly familiar with the physics and physiology of diving.

Air embolism may be a serious complication of scuba diving. A scuba diver should be well versed in the mechanism and avoidance of air embolism.

There may be a temporary loss of hearing when a person is subjected to pressure, but it usually subsides completely when the tissues of the middle ear have returned to normal.

Man's ability to adapt himself to a subaquatic environment by means of proper equipment, proper training and proper physical status is demonstrated by the feats of underwater demolition teams, of divers such as those at Weeki Wachee Spring, and expert skin and scuba divers throughout the world. Without such expert experience, instructions and equipment, man may well destroy himself.

Pain in the region of the ear and sinuses is probably man's greatest deterrent to the avocation of skin and scuba diving. The greatest dangers to man's subaquatic adventures are ignorance and foolhardiness. The person who is fearful of underwater and confined spaces should not and probably will not take up this avocation. Those persons who are poorly trained in the proper methods of skin and scuba diving and the limitations nature has placed upon them are apt to experience panic underwater and thus court disaster.

The otolaryngologic aspects of skin and scuba diving, notably aero-otitis media, aerosinusitis, air embolism and decompression sickness, are discussed. A study of a group of 38 divers is analyzed, and observations on divers at Weeki Wachee Spring are presented.

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Marshall Taylor Doctors Bldg.

COURS DE TECHNIQUE AUDIOMETRIQUE.

The Faculty of Medicine, Paris France, will hold a Course in Audiometry, October 12-18, 1959, under the chairmanship of Prof. M. Aubry of the Clinic of Oto-Rhino-Laryngology. The tentative program includes lectures on the scientific fundamentals, the technique of audiometry, discussions of the results and problems of interpretation, and round table discussions. Professor Aubry will be assisted by MM. les Professeurs Agreges J. J. Debain and R. Maduro; les Doctears P. Aboulker, J. Bouche, P. Clerc, H. Henrot, R. Maspetion, J. Pialoux, J. Bouchet, M. Burgeat, L. Chevance; P.-L. Klotz, P. Robert, B. Vallancien; Mme. S. Borel-Maisonny; M. le Professeur A. Didier; MM. R. Chocholle, J.-E. Fournier, R. Lehmann and A. Molles. The complete program will be ready in June, 1959. For further details write Secretariet: Service Oto-Rhino-Laryngologique Pavillon Isambert, Hospital Lariboisiere. 2 Rue Ambrose-Pare, Paris 10°), France.

STAPES MOBILIZATION, A PROBLEM IN COCHLEAR HYDRODYNAMICS.*

Preliminary Report.

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Histological evidence concerning cochlear lesions after experimental stapedial fractures in cats, recently has been presented by Singleton.[7] According to a personal communication with H. Schuknecht,[6] from whose laboratory the above paper had issued, this finding was not a singular one. Whereas, in the first few animals the lesions were observed in the upper basal turn, corresponding to a frequency of approximately 3,000 cps, more recently damage has been found to extend well into the second turn, *i.e.*, to approximately 1,500 cps. The lesions resemble somewhat those seen after a moderate acoustic trauma: haircells missing, not overlarge stretches; rather occasional gaps alternating with small islands of apparently intact cells. For anatomical details reference must be made to the paper by Singleton.[7]

After first being informed of these findings, this author was immediately reminded of observations made accidentally, but not infrequently, in his studies on cochlear models:[8] when either of the two cochlear windows was displaced inadvertently, a rather pronounced displacement of the cochlear partition ensued in its middle to lower portion; therefore, it seemed plausible to assume that the lesions observed by Singleton had been caused by stapedial displacements incurred during the manipulations at the stapes.

In order to obtain some preliminary information on this

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subject, slow-motion pictures (64 fr/sec.) were taken of the basilar membrane in a cochlear model. These models, five times the size of a human cochlea, have been described in detail in earlier papers.[3] While the pictures were being taken, the "oval window" was repeatedly depressed with a finger and released either slowly or suddenly. After slow depression or release, momentary displacement of the basilar membrane was seen to occur, mainly within the apical portion of the model; the bulk of the displaced fluids being exchanged through the helicotrema. If, however, the depression or, because it was technically simpler to execute, the release was carried out suddenly, the displacement of the basilar membrane extended well into its middle portion. The slow-motion pictures were not fast enough for an exact analysis of the pattern of these displacements; however, it is well known, from Bekesy's earliest experiments on the traveling-wave phenomena (1928),[2] that in case of a sudden stapedial displacement a single "bulge" is seen to travel along the entire basilar membrane from the window region toward the apex.

To understand these model phenomena properly, a few remarks may be in order about the physical properties of sudden shifts in displacement, commonly called *impulses*. Since they are not part of a continuous oscillatory event (otherwise known as "steady state" events), they are also called *transients* or *aperiodic* events. Such transients, an example of which is given in Figure 1, can be examined with the aid of a Fourier analysis with respect to their frequency spectrum, as first suggested by Lord Rayleigh.[5] Practical solutions were first presented by Kuepfmueller.[4] In contrast to steady-state events which, depending upon their complexity, contain one or several discrete frequencies, transients have a *continuous* spectrum. One might also say that the distribution of frequencies in such spectra is infinitely dense. One, therefore, speaks of *bandspectra* of a given *width*. The example of Figure 1 illustrates an instanteous change in displacement, an impulse of infinitely short duration. Its spectrum has an infinite width, although the amplitude per cycle is not uniform. It changes with frequency according to a hyperbola (see Fig. 1); however, impulses like that of Figure 1 hardly ever occur in mechanical systems. Usually, their time duration has

a well defined length. In that case the spectrum is also affected. There is a trading relation between bandwidth and time duration, so that longer time durations are associated with lesser bandwidths and vice versa; therefore, the spectra of the two impulses of Figure 2, with their gradual onset, have a limited bandwidth, all energy being confined to lower

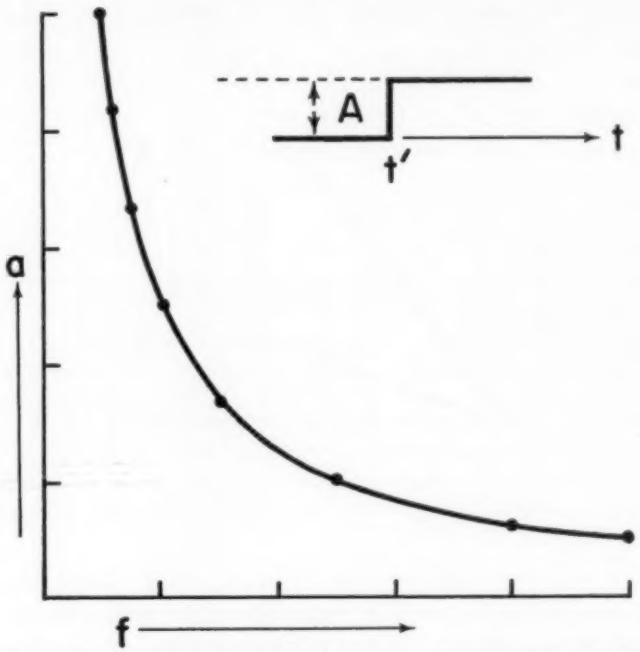

Fig. 1. Band-spectrum (amplitude/cycle) of a (hypothetical) instantaneous shift in displacement amplitude. The time course of the event is shown in the insert: At the time t' amplitude is instantaneously stepped up to the level A (adopted from[4]).

frequencies. This can be stated in a different form by saying that in case of an impulse starting gradually, the energy at a given frequency (which is much larger than one cycle) is less than it is in case of a sudden onset.

The significance of impulses can be illustrated with the aid

of a problem more familiar to otologists. Similar impulses, as shown in Figures 1 and 2, are produced when a tonal signal is suddenly switched on. At the onset of such a signal a click appears superimposed upon the tone. In the construction of audiometers it is desirable to eliminate such clicks. One achieves this simply by making the onset of the signal gradual (more like Fig. 2 than Fig. 1). The result is that the entire energy contained in the onset event is concentrated within the

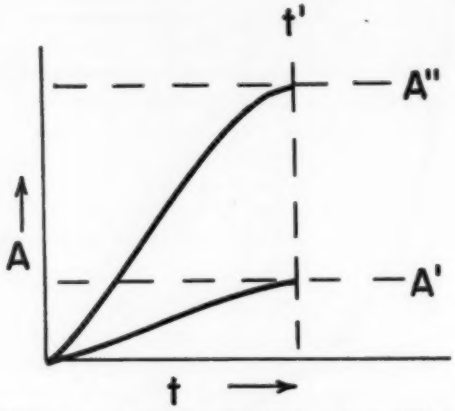

Fig. 2. Time course of two (hypothetical) gradual shifts in displacement amplitude. Both events are equal in time duration (t'), in the pattern of the change (s-shaped), and hence in their spectrum. One event (A') represents a low-level shift, the other one (A") a high-level shift.

low-frequency region where it remains *inaudible*, but the point to be made is that switching transients can never be *physically* eliminated.

Next, the displacement amplitude which might be incurred in stapedial manipulations, must be considered in terms of amplitudes of familiar sounds. Table I shows the amplitude of the human eardrum at the threshold of hearing within the ear's most sensitive range ($=10^{-9}$ cm.). From this value the stapedial amplitude has been calculated for the same condition; it is 2.5×10^{-10} cm., all values being given according to Bekesy's measurements.[3] Assumption of stapedial displacements during mobilization procedures of $\frac{1}{4}$ mm. ($=2.5 \times 10^{-2}$

cm.) are certainly not unreasonable. (Such a value may even be a little on the conservative side.) The db-ratio between both values cited is 160 db. According to what was said earlier about the spectra of impulses, this does not mean that during stapedial manipulations, amplitudes of 160 db are incurred with the frequency range of 1,500 cps to 3,000 cps,[1] where Singleton had found signs of cochlear damage.

Returning once more to Figure 2, where there are two impulses shown with the same gradual onset which is completed in the time t'. (There is a more precise way of expressing this by considering what is known as the *time constant*,

TABLE I.

Amplitude eardrum (threshold)	10^{-9} cm.
Amplitude stapes	2.5×10^{-10} cm.
Stapedial "rocking" of ¼ mm.	2.5×10^{-2} cm.
$R_{1.5}$: 160 db.	

(Amplitude data of Bekesy).

a technical term which will not be discussed here.) One of these curves represents a low-level shift in displacement of say 30 db. The other is much higher and might be 160 db, as in the present case, although such differences in magnitude cannot be drawn on the same linear scale: 30 times in one case and 100 million times in the other. The point to be made is that, although both impulses have an identical spectrum, the *absolute* amplitude at a given frequency is much higher in the larger impulse than it is in the smaller one. If one assumes the amplitude in the 1,500 cps to 3,000 cps range to be –30 db *re* total amplitude of the impulse, one finds in the first case (30 db shift) 0 db *re* threshold for that range, but in the second case (160 db shift) 130 db *re* threshold for the same range.

At this time, one can only guess as to the spectra which might be incurred in stapedial displacements during mobilization procedures. Certainly, during a slow and controlled rocking motion there is no energy at 1,500 cps to 3,000 cps; however, there are two other situations which need to be considered: first, when a surgeon realizes that he has suc-

ceeded in freeing the stapes and has inadvertently displaced it, he will most likely withdraw his instrument very abruptly and thus may introduce a sharp impulse; second, and that may be the more serious of the two situations: when the footplate or the fixing otosclerotic focus breaks it often does so with a snap. In that case motion cannot be controlled well enough to avoid a rapid and relatively large displacement because of the energy stored in the bone (and also in the instrument) prior to the snapping of the bone.

These preliminary considerations are still based upon parameters, many of which are rather vaguely defined. Before anything more definite can be said, more study is needed. For one thing, the mode of response of the cochlea (or the model) to sudden impulses was left out entirely from the present consideration.

SUMMARY.

An attempt was made on the basis of preliminary information to account for the observation of cochlear lesions in cats after experimental stapedial fracture. The attempted explanation for this observation was based upon considerations of principles of cochlear hydrodynamics.

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MODERN CONCEPT OF VESTIBULAR PHYSIOLOGY.*

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If we consider that it was just 85 years since Mach, Breuer and Crum-Brown, in 1874, located the sensations and reactions of rotation, on the semicircular canals, we must admit that this is a comparatively recent achievement, from a scientific point of view.

The labyrinth had not attracted much attention and previously, the investigations of Fleurens, cutting and severing the canals about 1824, were unnoticed or forgotten. The investigators of 1874 had known no other concept of the function of the canals than that they acted as fluid-rings, which were subjected to inertial forces in rotational head movements. It was not until 1896 (about 60 years ago) that the reactions of the semicircular canals were located in the endolymph and the sensory epithelium in the ampulla, as a result of the extensive and brilliant experimental work of Ewald.

At that time the existence of a cupula was denied, or it was regarded as an artifact. This statement was repeated even as late as 1931. The only possible way to stimulate the hair cells of the neuroepithelium of the crista was, therefore, supposed to be by bending of the hairs in one way or the other by the endolymph stream, caused by rotational head movements. Even for those who believed in the existence of a cupula, the bending of the hairs appeared still to be the important factor in the process of stimulation, even if this bending of the hairs was performed by action of the cupula. One difficulty was to explain how endolymph movements, which could not be very large, *i.e.*, no real "streaming" of endolymph, could be able to move the small cupula inside the wide ampullae of the canals. In microscopic sections the cupula appears always as a small appendix sitting on the

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crista. This is due to the abundant shrinking which this jelly-like structure suffers from the action of the fixing fluids. This was the situation until up to about 1930.

An important step was then taken by the use of Steinhausen's method of making the cupula visible, even in the living animal. This was possible by injecting into the endolymph a contrast medium such as India ink. (A short film was shown which demonstrated the actual shape and extension of the cupula when uninfluenced by any chemical methods for preparation of microscopic sections.)

From this it is evident that the cupula extends from the crista through the whole cross-section up to the vertex of the ampulla. It is also evident that every movement of the endolymph must, therefore, be transferred to the cupula. If the transmission of endolymph movements affecting a bending of the cupula has been clearly demonstrated by these experiments, there is still one important question to be settled: how can the energy of mechanical bending of the cupula be transformed into the electro-chemical effect of nerve stimulation?

From the short historical survey which I have tried to present, it might seem natural that the bending of the hairs of the hair cells should be regarded as the essential mechanism for transfer of mechanical energy to the sensorial cells. This is not only a labyrinthine problem; the same mechanism must be operating in stimulation of the hair cells of the cochlea and the maculae. In the semi-circular canals the endolymph is assumed to bend the cupula, and the cupula, the hairs. In the cochlea the basilar membrane is supposed to move up and down, and thereby producing radial movements of the upper surface of the organ of Corti with its hair cells. It is further supposed that the tectorial membrane rests loosely over the organ of Corti, that it should be relatively stiff so that the free endings of the hairs should be bent outwards and inwards when the hair cells of the organ of Corti are vibrated by the movements of the stapes.

The tectorial membrane, however, is of the same jelly-like nature as the cupula, and it seems to me rather strange to suppose that this jelly should have a rigidity greater than the moving cochlear partition of basilar membrane and cells.

Further, it is beyond doubt that the histological picture of the tectorial membrane, as a free floating formation over the organ of Corti, attached only to the limbus is an artifact, due to the shrinking agents necessary for the fixing and preparation of the histological sections, just as the cupula always shrinks to only a part of its natural size as seen in all microscopic sections.

It has been shown by Anderson Hilding, in fresh preparations, and in frozen dried sections by deVries, that the tectorial membrane is fixed on either side of the organ of Corti, which means that the tectorial membrane is attached to the whole upper surface of the organ of Corti with its hair cells and hairs. This is easily proven by anybody who can use a dissecting microscope and a preparation needle.

In 1954 Vilstrup and Jensen showed the presence of hyaluranic acid in the endolymph of Acantias, and Jensen, Koefoed and Vilstrup showed that potential changes could be produced in hyaluranate solutions on displacement of the fluid. Vilstrup suggested that these "flow potentials" could be of importance for the stimulation of the sense organs of the ear.

As is well known, electrical potentials can be led off from the cochlea, potential changes, synchronous with the sound waves of tones transmitted to the ear, can be recorded. This is what is meant by the Wever and Bray effect, or cochlear microphonics. If the basilar membrane and the organ of Corti is pushed upwards from the scala tympani, this gives a potential change in the negative direction which lasts as long as the displacement lasts, and if the movement is performed in the opposite direction this will give an increased positivity. This apparently means that a displacement gives a potential change of a D.C. type, and that rapid changes of displacement gives an A.C. response. Exactly the same happens if the electrical changes are led off the ampulla. If the cupula is moved in one direction one change can be recorded, *e.g.*, in a positive direction; if the cupula is bent in the opposite direction, the potential change is reversed, and further, rapid potential changes can be recorded if the cupula is exposed to a tone stimulus when a fistula is made in the canal.

How can these facts be brought in accordance with the historical conception of a bending of the hairs as the mechanism for transmitting of the mechanical movements into these known electrical effects? Is there any example of a living tissue which can give an effect like this, giving a positive potential change in one direction when bending a hair, and negative when bending the same hair in the opposite direction, or is it even probable?

It is known that the potential changes of the cochlear microphonics are sensitive to oxygen deficieney and to certain poisonous substances which are supposed to be transmitted to the hair cells from some secretional epithelium over the endolymph; therefore, it seemed pertinent to study the mechanism of endolymph secretion to see whether some information could be obtained which would be of value in the interpretation of the mechanism of stimulation.

For several reasons it seems probable that mucopolysaccharides played an important role in this metabolism. The radioactive isotope of sulphur S.35 was, therefore, chosen as a tracer. We have used pigeons for these experiments.

To be able to explain the results of our experiments, I must explain the technique. The labelled isotopes are injected intramuscularly, the birds are killed after different periods of time from one hour to 30 days. Serial sections are prepared from the inner ear. These microscopic sections are covered in the dark with a layer of photographic film. The sections are then stored, still in the dark, for some length of time, thus exposing the film to the radiation from the radioactive parts of the tissues. When developed the film showed the exact distribution of the sulphur compounds, due to the metabolic processes. The results of these investigations will be apparent from the slides. The first slide is a cross-section of the ampulla from a pigeon killed four hours after the injection. It shows exposed silver grains over the epithelium of the sidewalls of the ampulla, the region which is called planum semilunatum.

If we look at this epithelium in pigeons kept alive for different periods after injection, we can ascertain the time required for the labelled molecules to participate in the metabolic

processes and pass through the different tissues and fluids of the inner ear. After one hour these can be seen in the film as a few scattered silver grains.

After two hours there are more silver grains in the epithelium, and after four hours the irradiation has produced a very dense black ribbon over the epithelium and nowhere else to the same extent.

After six hours there is still a dense blackening; after 18 hours the intensity is slightly decreasing. After 24 hours there is only an insignificant irradiation, which means that all the labelled sulphur has passed through the epithelium into the endolymph cavity.

The shrunken cupula shows no irradiation after one hour nor after two hours. The irradiation from the cupula is insignificant after four hours, slightly greater after six hours. At 24 hours, at the time when most of the radioactive sulphur has left the epithelium, the irradiation from the cupula is dense, as are also the clouds of coagulated endolymph which can be seen inside the membranous canal. After three or four days the irradiation from the cupula and endolymph again decreases.

We have followed the fate of this radioactive compound up to 30 days after injection. This investigation shows that the radioactivity in the clouds of coagulated endolymph decrease, and disappear after five to ten days; that the activity in the cupula also decreases but can still be observed to some extent, even after 30 days; further, it is possible to follow the excretion of the radioactive granules, which definitely pass through the ductus endolymphaticus (S. Guild) are condensed there into a thrombus-like coagulum and pass through the saccus endolymphaticus into the loose connective tissue surrounding this tube, and apparently are excreted into the cerebrospinal fluid through a structure with finger-like processes resembling the chorioid plexus in the ventricles of the brain.

Expressed in chemical phraseology, this would means that the secretory epithelium builds up and secretes sulphur-bound compounds, which, when secreted by an epithelium like this, should be sulpho-muco-polysaccharides. In order to confirm

this assumption, and to investigate which compounds are coupled to the sulphur, together with Dr. H. Bostrom, I have collected endolymph from pigeons injected with S.35 and by means of electrophoresis and chromotography, examined the chemical content of the fluid. So far this has only confirmed that the organic compounds are protein-bound sulpho-mucopolysaccharides, but further investigations are planned.

Anatomically, the distribution of the secretory epithelium has been found to embrace the cupula from both sides, and to continue on the utricular as well as the canalicular side, thus totally enclosing the neuroepithelium with its cupula.

From a functional point of view it would be astonishing if the secretion of such complex compounds into the cupula and then into the endolymph, were not of some fundamental significance to the physiological function of the mechanism located in the ampulla.

To discuss this fundamental question we must consider some facts about the cupula. It is generally assumed that the cupula is built up as a meshwork or sponge. It is apparently in this meshwork that one finds the mucopolysaccharides. It is also generally accepted that the hairs of the hair cells lie in fine canals in the cupula substance.

If a very small drop of a dye is deposited in the cupula it is possible to see the dye particles moving longitudinally up and down, if the cupula is moved by some external force.

Accordingly, these two observations would suggest that bending or pressure on the cupula could produce fluid movements in the cupula meshwork and in the fine canals around the hairs of the hair cells. With regard to the shape of the cupula fixed to the surface of the crista, as it is, one would have reason to expect the fluid in these small canals to move in one direction with an ampullo-fugal displacement of the cupula, and in the opposite direction with an ampullo-petal deflection of the cupula. This is an assumption, and I will leave it as such at the present moment.

If the movements of the cupula at the threshold must be extensive before the neuroepithelium could react, then this would speak in favor of some mechanism which could bend or tear the hairs or even deform the cells. If, on the other

hand, the movements were very small, such a mechanical stimulation of the hair cells would be impossible, and the assumed fluid movements in the cupula could then be of greater importance in the explanation of those phenomena in the inner ear to which we will refer later. An experiment was made to determine the cupula deflections due to forced fluid movements through a fine canula cemented into an opening in the canal wall. This experiment showed that even with movements which could give a deflection of at the most, half a degree of arc, unmistakable vestibular reactions were recorded. This would mean that the tip of the hair could not have been moved more than 0.3 μ, probably less.

Turning then to the hair cell, it is known from the electron-microscopic studies of these cells by Wersall, Catherine Smith and others that there are about 50 to 60 hairs extending from each cell; they are about 40 μ long and 0.2 μ in diameter. They have a central fibrillary structure only in the basal part of about 4 μ in length. These 4 μ in the basal part of the hair could theoretically be regarded as a stiff part which possibly could react to a bending, whereas the distal 9/10 of the hair would be totally flexible; furthermore, the basal part of the hair just passes through the cuticle of the cell, but never shows any continuation down into the cytoplasm of the cell. Morphologically, therefore, this picture does not seem to support the assumption of a stiff stricture which would be able to stimulate the cell by bending of the hair.

According to the electron-microscopic picture, the hair seems thus to be built up of a finely granulated protoplasma with a thin outer plasma membrane, and that this membrane is the continuation of the plasma membrane of the cell; therefore, the hairs of the hair cells must be regarded as long, tube-like extensions of the cell-surface, giving a considerably enlarged surface area of the cell. The cell surface (except hairs) can be estimated at about 40 to 50 μ^2 but with the surface of the hairs the total area will amount to between 3,000 and 4,000 μ^2. This seems to be a very important fact, because if we consider that these hairs protrude out into the cupular substance lying in fine canals, and that this cupular meshwork is soaked through with a secretion containing two important constituent parts, the sulpho-mucopoly- sac-

charides and potassium, then I believe that we have the right to presume that this is an arrangement which can be described as, *1.* an insulated entity of electrically leading solutions, the cell; *2.* this cell sends out a great number of antennas, the hairs, into a *3.* highly charged electrical field—the solution in the cupular meshwork.

We know that in a solution of mucopolysaccharides and electrolytes, the very large and highly negatively-charged molecules of the polysaccharides are binding the positive electrolyte-ions of potassium to its surface. This means that as long as no physical changes are imposed on this mechanism, the positive and negative charges are kept in balance; however, as soon as movements are imposed on the fluid, the positive potassium-ions, which are more movable, will move in the direction of the fluid displacement and leave the slow polysaccharide molecules behind. This means that the antennas of the hair cells, surrounded as they are by these molecules and moving ions, are, on their surfaces, exposed to the electrical charges which are predominant at that moment. Moving the fluid then in a direction toward the hair would mean an increased positive; a movement in the opposite direction, increased negative. We also know that the potassium content in the endolymph is about the same as in the interior of the cell, and that the endolymph is positive with regard to the potential of the hair cells. This high concentration of potassium and the high potential difference between the outside and inside of the hairs and the cell is a very remarkable fact. If a potential change at the cell's or hair's plasma membrane should give an increased permeability of that membrane, it would be totally disastrous to the cell, because if the positivity is increased outside the cell this cell would immediately be overloaded with potassium, to an extent inconsistent with life. Second, it would be impossible for the cell to work against the high concentration gradient of potassium and against the electrical gradient between cell and endolymph even if the electrical charge is slightly lowered or raised around the hairs. Already, from the fact that these two gradients are a constantly present potential danger to the cell we might presume that the plasma membrane that shields the cell from the

surrounding fluid in the cupula is *impermeable*. This is not a common thing, but is by no means unique.

This implies that the cell works as a condenser; a positive charge applied outside the insulating membrane gives a negativity to the inside salt solutions of the cell protoplasm, and vice versa.

This might further explain the facts of a standing and remaining potential value in the cell due to a certain mechanical displacement, *e.g.*, of the cupula or the basilar membrane, and it explains the rapid potential changes like those of the cochlear potentials, and above all, makes it possible to explain a function capable of moving in both directions, positive and negative, from the position of a resting cell-potential. This has not been possible to explain through the hypothesis of the bending hairs. This might also well fit in with some other known investigational results.

For instance the observations on the cochlea, by Hallowell Davis and his collaborators, by Békésy and others, have indicated that the source of the potential changes due to the mechanical stimulation is at the hair-bearing end of the hair cells and orientated perpendicularly to the cell surface. This cannot possibly be explained in any other way than through a condensor effect at the cell membrane of the hair cells.

Further, there is another experiment of Davis, et al., which is of interest in this connection. Recording the cochlear microphonics through a microelectrode, which was slowly moved from the endolymphatic cavity in the cochlear duct into a hair cell in the organ of Corti, Hallowell Davis could show that in the moment the electrode passed through the cell membrane which was proven by the sudden potential drop from about +60-90 mv to about –70 mv, there was an equally sudden phase shift in the electrical response to the sound-waves of the test tone. This means that when the mechanical movements of the cochlear fluids by an inward movement of the stapes, gave a potential change in the positive direction, this then became negative in the cell, and vice versa, a condensor effect.

Considering the situation in the cochlea, we learn from every textbook that the movements of the basilar membrane

are of prime importance for the function of the cochlea, and that these movements are transformed into rocking movements of the organ of Corti, which in this way rubs its surface against the supposedly stiff tectorial membrane, thus explaining the bending of the hairs of the hair cells.

It has been pointed out that the first condition for a transformation of the up-and-down movements of the basilar membrane into movements in a radial direction is that the inner pillar has a firm support on the tip of the bony spiral lamina. This is the case in the guinea pig, but not in man. In the human ear the organ of Corti is situated on the cochlear partition halfway between its two supporting points at the spiral lamina and the spiral ligament. The result is that the movements of the hair cells can be expected to occur only in the directions up-and-down, and only to a very small extent in any other direction. These movements could not possibly promote bending of the hairs in the traditional way of thinking. Besides these objections, I would remind you of the calculations of Békésy regarding the magnitude of the movement of the basilar membrane, which at threshold level could not be expected to exceed the diameter of a hydrogen atom.

This microscopic section of the pigeon's cochlea, shows the hair cells supported by a massive cushion of connective tissue which does not facilitate a vibration in the traditional sense, and still I believe that the cochlea of the pigeon, as well as that of man, is the organ of hearing; therefore, I think that it is much more important to note what actually can happen to the tectorial membrane, than to the movements of the basilar membrane. There is no doubt that the basilar membrane can move and that it moves in the way Békésy has so beautifully shown in his experiments, but I am quite convinced that this is of secondary importance to the movements in the substance of the tectorial membrane with the possibilities this can give for rapid changes in electrical potentials at the surface of the hairs.

Nobody has suggested that the crista in the semicircular canals should be the moving part against a stiff cupula, or explained the stimulation by rotational head movements, or

explained the response to tones in the Tullio-fistular-experiment; therefore, I believe that it is necessary to judge the function of these very important jelly-like structures from a unitary point of view; the manner in which the cupula functions in the semicircular canal in response to endolymph movements, as well as to tones, cannot be fundamentally different from the functional mechanism of the tectorial membrane or the otolith membranes. With small differences according to their use for special mechanical purposes, it is most likely that they function in principally the same way.

In conclusion, I confess my belief in this situation of conflict with current doctrines, which I have deliberately been stirring up.

I think I would rather believe that some little devil, sitting on the crista, directs the stimulation one way or another, than to believe that a bending of a hair would be able to produce a cell stimulation in one direction and that the bending of the same hair in the opposite direction could give the opposite response, unless somebody could explain to me this miraculous phenomenon in plain words based on known and undisputable facts.

What I really believe is that, whatever mechanism might be operating, it must have the ability both to increase and to decrease the cell potential of the hair cell. Taking into consideration the electrical possibilities of the endolymph solutions—mucopolysaccharides and potassium—I believe that they are most likely to be the source of electrical changes, working on the membrane of the hairs and cells the same way as a condensor works.

IMPROVEMENT OF SOUND TRANSMISSION IN THE FENESTRATED EAR BY THE USE OF SHEARING FORCES.*†‡

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and
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Earlier studies of the sound transmission from the ear canal to the organ of Corti made it clear that the transmission is done in two separate steps. In the first step the vibrations of the air are transmitted to the perilymph, a fluid. To avoid energy loss during this transmission the well-known middle ear mechanism is used as a transformer to match the air-fluid boundary. In the second step, the movements of the perilymph are transmitted to the cells of the organ of Corti, a more-or-less solid material. To avoid the energy loss in this transmission from a fluid to an elastic membrane a second transformer is used. The first mentioned transformer in the middle ear is of the type of the hydraulic press and is well known. The second transformer in the organ of Corti, however, is something new. It is a transformer based on shearing forces described by Bekesy in the *Journal of the Acoustical Society of America.* This new finding by Bekesy naturally gave rise to the following question: "Can a shearing force transformer be used to improve, in the fenestrated ear, the transmission of the air-borne sound to the newly created covered extratympanic vestibular fenestra?"

How a shearing transformer acts may be seen in the upper drawing of Fig. 1. This figure shows the cross-section of a membrane stretched across the edges of an open tube. In the middle of the membrane, a small single force is applied as illustrated by the small arrow. As a consequence of this

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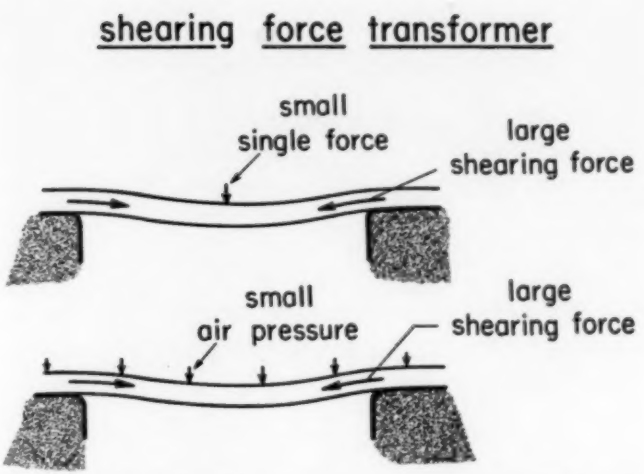

Fig. 1. The upper figure shows that a small single vertical force can produce a large horizontal shearing force. The lower figure shows the same action for a small air-pressure.

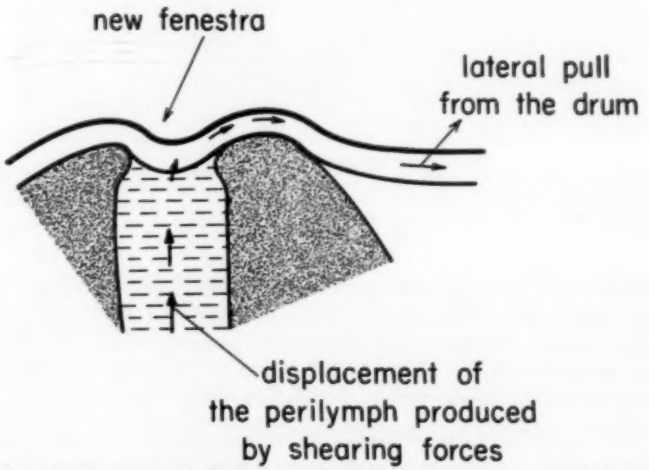

Fig. 2. Demonstrates how the large lateral shearing force displaces the perilymph below the fenestra.

Fig. 3. The incus has been removed. The tympanomeatal flap has been created. Exposed to view are the newly created fenestra in the surgical dome of the vestibule, the tympanic course of the facial nerve canal, the head and neck of the malleus with its ligamental attachments, the chorda tympani nerve and the head and crura of the stapes.

small force, considerable stress is developed on the edges of the membrane. This force is larger than the force applied in the center; therefore, the system represents a transformer for forces which increases a small force to a larger one similar to a lever.

The lower surface of the membrane is attached to the edge of the tube (as seen in the drawing) and only the upper surface of the membrane will be displaced in a gliding movement

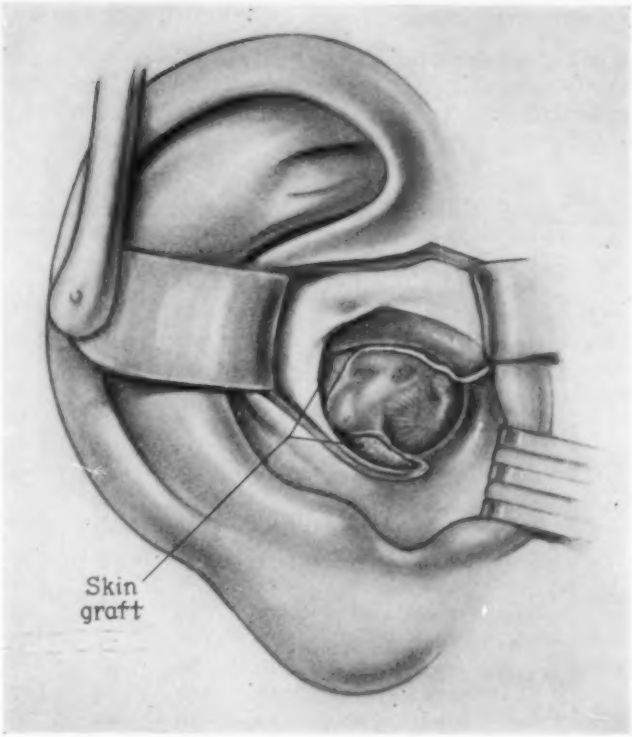

Fig. 4. The tympanomeatal flap can be seen covering the head and neck of the malleus, sealing the tympanic cavity and covering the newly created fenestra.

relative to the attached lower surface. This gliding motion of the upper surface in relation to the fixed lower surface is called "shearing." Depending on the thickness of the membrane the forces produced on the surface are somewhat smaller than the entire lateral pull on the edges of the tube, but they may act as a force increase in relation to the central force.

Not only a single central force but also an evenly distributed pressure on the entire upper surface of the membrane pro-

duces a strong lateral pull and a corresponding shearing force on the upper surface of the membrane. The action of the air pressure is shown in the lower drawing of Fig. 1.

Fig. 2 shows how the lateral pull on the edge of the membrane is transmitted by shearing forces to the perilymph in the newly formed fenestra. As can be seen, a section of the

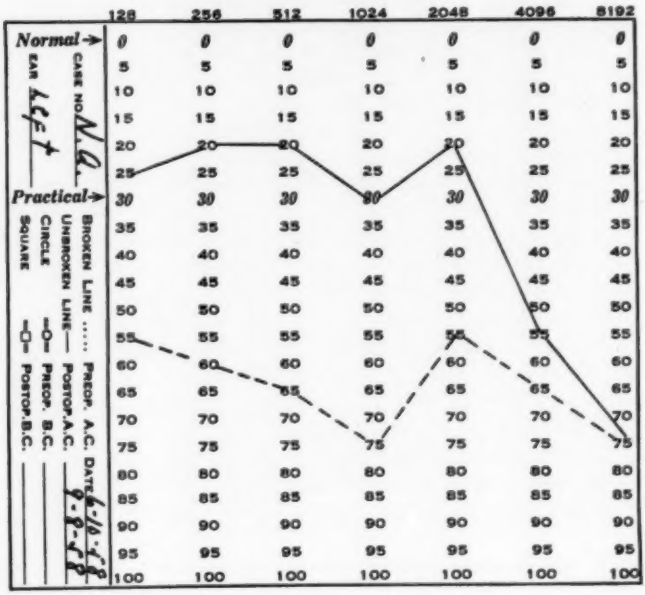

Fig. 5.

lower surface of the membrane is firmly attached to the bony wall of the canal; but the upper surface of the membrane glides freely over the bony edges and will transmit a pulling force on the window membrane if it is properly invaginated.

Here again the thickness of the flap, the size of the window, the length of the attached section of the flap, tension of the eardrum, etc., all play an important role in determining how

the lateral pull of the eardrum is transmitted to the perilymph, produced by the force transformation, as shown in Fig. 1.

If the application of the shearing force transformer is successful and functions as a transformer, then the force acting on the perilymph as a consequence of the shearing force is larger than the simultaneously-present sound pressure on

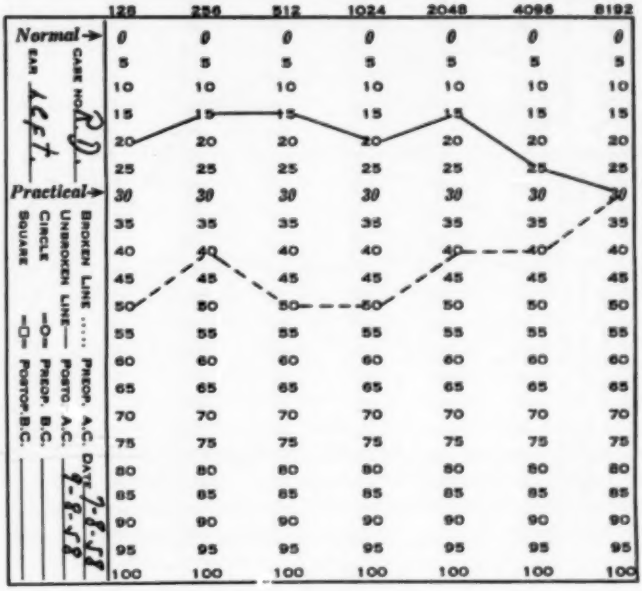

Fig. 6.

the window. In this case phase relations between the direct sound pressure on the window and the shearing force produced by the sound pressure on the eardrum are not important when we add the two tones together.

Experiments on models and on the operating table have shown that a shearing force transformer can be a very useful method. Since the physical part of the method seemed to promise the desired results, we are now working on the biolog-

ical side of the question; namely, how the shearing force transformer should be constructed from the living tissues of the middle ear so that the mechanical construction fits most naturally the biological conditions of the human middle ear. We think that in general this problem has been basically

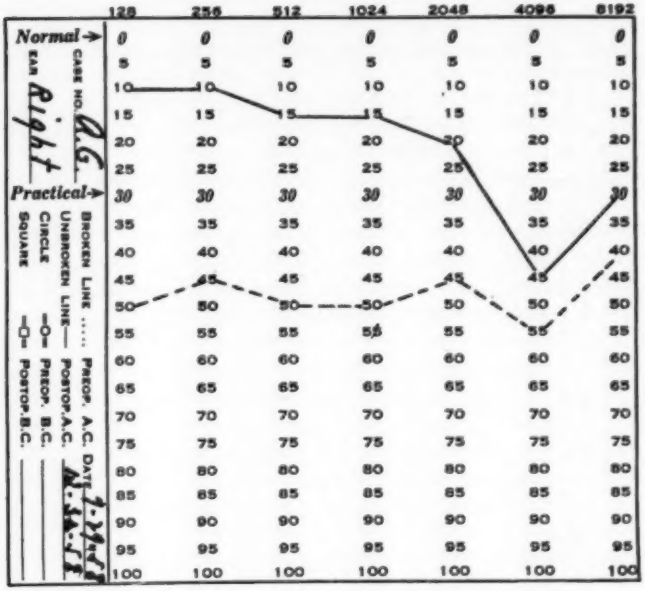

Fig. 7.

solved with the use of the malleus and the tympanomeatal flap for creating the shearing force transformer.

Figs. 3 and 4 show the technique of creating the shearing force transformer (see Figs. 5, 6, 7, 8).

We shall report the technique minutiae after we have had sufficient time to follow up the patients. Then we would also like to report, in greater detail, the physical principles of the shearing force transformer upon which this new technique is

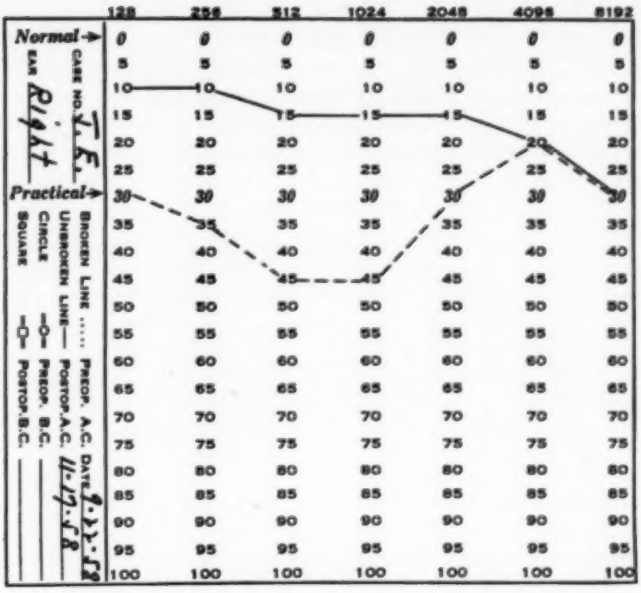

Fig. 8.

based, as well as the specific method of approach to the problem of deliberately and consecutively improving the hearing to the near normal and, if possible, to the normal level, following the fenestra nov-ovalis operation.

ELEVENTH CONGRESS OF THE INTERNATIONAL ASSOCIATION OF LOGOPEDICS AND PHONIATRICS.

Those unable to attend this Congress but who would like to secure a copy of the Report of the Proceedings can obtain it at the reduced rate of *seventeen shilling and sixpence* (17/6) by ordering it in advance, enclosing payment, from the Secretary, before August, 1959. Secretary's address: Peggy Carter, L.C.S.T., 46, Canonbury Square, London, N. 1.

CRURAL REPOSITIONING IN STAPES MOBILIZATION SURGERY.*†

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INTRODUCTION.

Crural otosclerosis is, in my opinion, the greatest cause for failure and refixation after total mobilization of the stapes by the Rosen[1] or other[2,3,4,5,6,7] indirect or direct surgical attacks upon the stapes. Fowler[9] has made a great contribution to the lasting results of stapes surgery by removal of the anterior crus and mobilization of the posterior footplate and crus. This anterior crurotomy has given us the greatest per cent of cases with complete closure of the air-bone gap, and of lasting hearing improvement in anterior crural otosclerosis. There remains, however, the problem of the surgical attack when the otosclerotic lesion involves both crura. In bi-crural otosclerosis, I have had significant success with central repositioning of the posterior crus, accompanied by anterior crurotomy, tendonotomy and central footplate mobilization.

In a review of our photo-pathological slides on the stapes, Farrior[10] reveals that about 75 per cent of cases have otosclerotic lesions localized to the anterior footplate or anterior crus. These respond to total stapes mobilization or to anterior crurotomy. There are about 20 per cent of the cases with bi-crural otosclerosis, and an additional 5 per cent with more extensive disease showing complete involvement of the footplate or obliterating lesions of the oval window niche. This paper will deal with that most interesting 20 per cent of the cases which have bi-crural otosclerosis.

With the total mobilization technique, we initially obtained

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only 18 per cent of successful results in these cases of bi-crural otosclerosis, as compared to 89 per cent with anterior footplate otosclerosis. In a recent 100 mobilizations, we have performed central repositioning of the footplate in 19 cases with 80 per cent of these obtaining a worthwhile hearing improvement at the one-month audiogram. In many of these

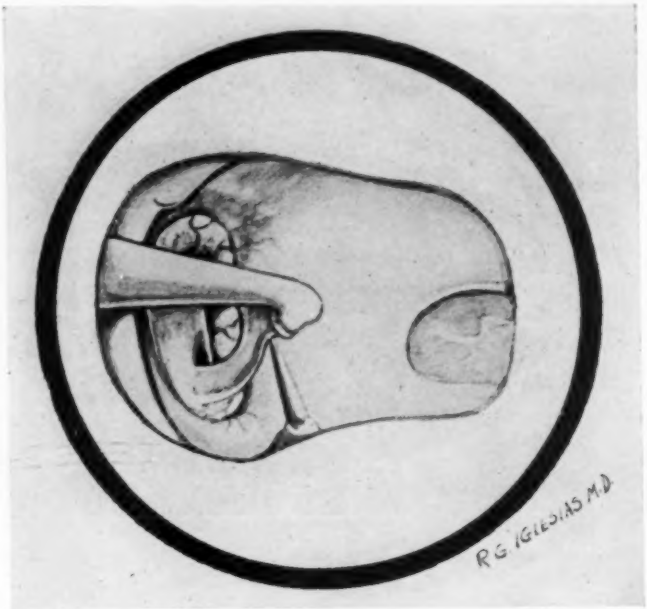

Fig. 1. Crural otosclerosis is the greatest cause of refixation after total stapes mobilization. Bi-crural otosclerosis occurs in about 20 per cent of cases and requires special surgical technique; for lasting results crural repositioning is advocated.

cases, the otosclerosis was so extensive as to be considered almost inoperable. This improvement of results in cases of bi-crural otosclerosis has encouraged me to make this preliminary report on crural repositioning. Dr. Bruce Proctor[12] has encouraged me to make this report, stating that he knows of no similar technique reported in the literature.

SURGICAL TECHNIQUE.

Adequate exposure of the stapes and stapedius tendon is obtained through the exposure of the operator's choice (see Fig. 1). A section of the stapedius tendon is removed for accurate microscopic diagnosis of the posterior crural otosclerosis and to obtain greater mobility of the stapes (see Fig. 2-a). Having established the diagnosis of bi-crural

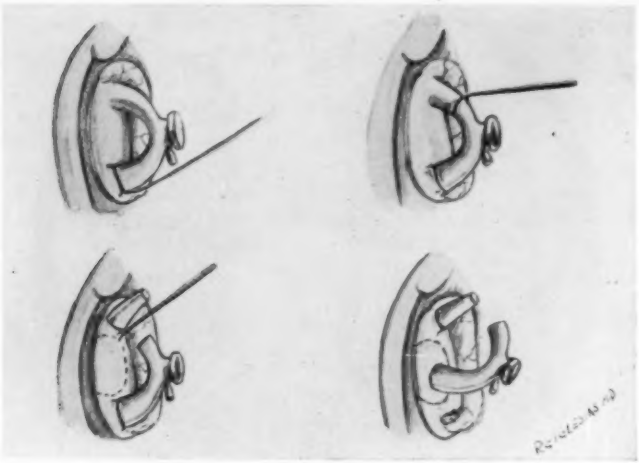

Fig. 2. Crural repositioning has given us improved results in bi-crural otosclerosis. The steps in surgical technique are: 1—2-a. Tendonotomy and posterior crural-lysis; 2—2-b. Anterior crurotomy; 3—2-c. Central mobilization of the uninvolved footplate; 4—2-d. Central repositioning of the posterior crus.

otosclerosis with a reasonable central area of normal or near-normal footplate, I then proceed with the stapes surgery by fracturing the posterior crus away from the otosclerosis (see Fig 2-a); second, an anterior crurotomy is performed (see Fig. 2-b); third, the central area of the footplate is mobilized (see Fig. 2-c), and fourth, the posterior crus is moved forward to approximate this central area of mobilized footplate (see Figs. 2-d; 3).

TENDONOTOMY.

The section of stapedious tendon is usually removed with the Bellucci scissors (see Fig. 2-a). If the exposure or the length of the tendon does not permit the use of the scissors, the tendon is sectioned with the sickle knife and the eminence removed with the curette. It is important to remove a section

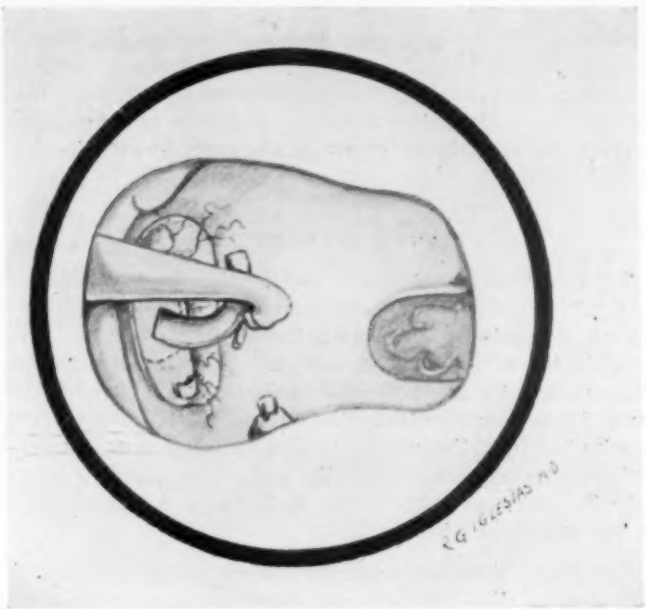

Fig. 3. The completed posterior crural repositioning demonstrates the posterior crus in position over the mobilized impacted central area of the footplate to which it becomes adhered by fibrous union.

of the tendon to prevent postoperative fibrous union and subsequent limitation of the mobility of the stapes.

POSTERIOR CRURAL FRACTURE.

The posterior crus is then fractured away from the otosclerosis as near as possible to the footplate, in order to obtain

the longest available section of the posterior crus. Posterior crural-lysis is performed by wedging a pick or chisel between the posterior-inferior margin of the oval window and the posterior crus. It is important to fracture the posterior crus before performing the anterior crurotomy, otherwise the posterior crus may fracture near the neck and be too short. If there is some doubt about the diagnosis of the posterior-crural otosclerosis, the efforts about the posterior crus are directed first toward an attempt to mobilize the posterior half of the footplate. If the posterior crus is found to be free, this mobilization is continued, and subsequently the anterior crurotomy is performed. In anterior-crural otosclerosis, Shea[13] prefers to mobilize the posterior footplate and crus before performing the anterior crurotomy in order to avoid posterior crural fracture.

ANTERIOR CRUROTOMY.

The anterior crurotomy is one of the most valuable and difficult techniques in all of stapes surgery. I usually approach the anterior crus with the chisel or gouge. Where possible, the curved chisel or gouge is inserted over the top of the incus and the force directed in the long axis of the anterior crus. By scraping on the anterior crus, the shaving technique usually sections the crus. If the anterior crus cannot be sectioned by manual control, the end of the chisel is touched with the micro-vibrating sides of the forked end of the 256 or 512 magnesium tuning fork.

Sometimes it is necessary to approach the anterior crus from behind the incus, under which circumstances similar maneuvers are directed toward sectioning the anterior crus.

The stump of the anterior crus is then folded forward with a right angle pick. This right angle pick also serves to make certain that a portion of the anterior crus has been removed. Fowler advocates removal of a section of the crus to prevent post-operative reunion.

FOOTPLATE SCARIFICATION.

The central portion of the footplate is first scarified to en-

courage postoperative fibrous union with the cut end of the posterior crus.

FOOTPLATE MOBILIZATION.

In bi-crural otosclerosis, the anterior, posterior and inferior margins of the footplate are usually fixed in the otosclerotic process, (superior bi-crural otosclerosis is similar but rare, less than 1 per cent). There is usually a central area of normal or near-normal footplate. This central area of the footplate is mobilized with the pick or chisel. I prefer to impact this central area in order to insure a freely movable non-bony healing of the margins of the mobilized trap door in the footplate.

CRURAL REPOSITIONING.

The freely movable posterior crus is then swung forward to approximate this mobilized central area of the footplate (see Figs. 2-d; 3). The freely movable posterior crus will usually maintain its position in contact with the central area of mobilized footplate. This contact is encouraged by gentle pressure on the incus. In rare circumstances, it has been necessary to insert gelfoam behind the crus to maintain this central repositioning of the posterior crus.

In only one instance has the anterior crus been longer than the posterior crus. In this instance, the anterior crus was repositioned in a similar manner.

ARTIFICIAL CRURA.

At one time I made the statement that "bi-crural otosclerosis is beyond the limits of present day mobilization surgery" (Farrior[14]). From this point I began to develop the above-described technique. This technique was at first utilized only in the far advanced cases which were beyond the limits of fenestration surgery. I now utilize, or attempt to utilize, this technique in cases of bi-crural otosclerosis with a wide oval window niche.

Central repositioning at the posterior crus is, to me, preferred to the use of the artificial crura, simply because I do

not like foreign bodies in the ear. Artificial crura may be indicated in extensive involvement of the crura where there is a reasonable area of normal or near-normal footplate. I believe that artificial cruralization is to be preferred to solo footplate mobilization or simple a-crural footplate perforation (Rosen[15]). Artificial crura may also be indicated where there is marked otosclerotic narrowing of a deep oval window niche despite a relatively wide normal area of deep blue footplate. This, to me, is the one specific indication for Schuknecht's[16] stainless steel cruralization. I feel that both central repositioning of the crura and the use of artificial crura are limited to that 20 per cent of cases which show bi-crural otosclerosis, and possibly to some reoperations on the stapes in which the perfectly operable, ideal stapes has been violated by inadequate or inappropriate primary stapes surgery.

FOOTPLATE EXCAVATION AND OVAL WINDOW GRAFTS.

Up to this point we have been discussing surgery on those stapes which present a reasonable area of normal or near-normal stapedial footplate and bi-crural otosclerosis. In these cases, I prefer the central repositioning of the posterior crus as a first choice, and as a second choice the use of the steel or polyethylene crura. I do not fenestrate or excavate this oval window niche, as I like to have the central area of mobilized footplate protecting the inner ear. I do not feel that making windows in the footplate is indicated when there is a reasonable area of normal or near-normal footplate.

Excavation of the footplate and the application of an oval window graft may prove to be indicated in those cases where there is complete otosclerotic involvement of the footplate. Shea[17] has had some dramatic success with the excavation of the footplate and the application of the vein graft. I feel that this surgery has a role in the field of stapes surgery; however, I feel that it should be limited to those cases where there is complete otosclerotic involvement of the footplate.

Complete otosclerotic involvement of the footplate usually manifests istelf clinically by a very wide air-bone gap, and is clinically the most suitable case for the fenestration opera-

tion. Having had reasonable experience (Farrior[18,19,20,21]) with the modified Lempert[22] fenestration operation, I usually advise the fenestration, in clinically suitable cases, in the presence of complete footplate otosclerosis or in the presence of obliteration of the oval window niche, because the fenestration is the most predictable operation on the ear (Shambaugh[23]).

HISTORICAL.

My first case of crural repositioning, September 10, 1957, No. 269, was performed on a class-D case, a 54-year-old judge with an 85 db hearing loss and a bone conduction loss of 42 db, a case on which I would not do a fenestration. Examination of the stapes under the microscope revealed complete otosclerotic fixation of both crura. In order to investigate the condition of the footplate, I fractured the crura free and moved them to one side. This revealed a small central area of normal footplate. This central area of footplate was mobilized, and the posterior crus repositioned over it. I am not certain whether this trap-door in the footplate is fixed to the crus by a bony or a fibrous union, for the remaining posterior crus was very short; nevertheless, for the past 20 months, this patient has maintained a 43 db hearing improvement to the 42 db level. There are a number of other cases of crural repositioning which have maintained excellent hearing improvement for more than one year. In these cases, with the dissolution of the ossicular chain, the patient may obtain a significant hearing improvement in the operating room, but it is not the complete and dramatic improvement which occurs with an intact ossicular chain. About four weeks must be allowed for union between the base of the posterior crus and the freely movable impacted central area of the footplate. These patients may notice wide fluctuations in their hearing for the first month, but thereafter the hearing stabilizes.

Analysis of results for the scientific exhibit presented at the A.M.A. revealed that in bi-crural otosclerosis we had obtained only 18 per cent results. From that time, May, 1958, crural repositioning was regularly employed with this standardized technique in the 19 cases of bi-crural otosclerosis

which occurred in the 100 stapes mobilizations (371 to 470) in the period from May to December, 1958. These cases are selected for audio-analysis.

PREOPERATIVE AUDIOGRAMS.

I have long held the opinion that bi-crural otosclerosis is indicated clinically by a sloping audiogram (Farrior[14]). A review of these audiograms indicates that 84 per cent showed a gradual downward slope from the low to the high tones, with a greater than 10 db drop. At this time I am undecided as to which is of the greatest clinical significance, the retained hearing for the low tones or the loss of hearing for the high tones; actually, I believe that the retained hearing for the low tones is indicative of a small area of normal or near-normal footplate, which is freely movable on the superior margin.

Only one case (5 per cent) presented a rising audiogram. A rising audiogram usually indicates a freely movable posterior half of the footplate, and it may be that in this particular case I may have made an error in diagnosis of the posterior crural involvement.

Only one case (5 per cent) presented a flat audiogram. A very flat audiogram with the wide air-bone gap of greater than 40 db is, to me, clinically indicative of complete footplate otosclerosis. It may be that in this case the patient actually had complete footplate otosclerosis which was under-diagnosed. In this case, No. 374, the stapes mobilization was a failure, and we have subsequently done a fenestration operation. In the course of the fenestration operation, No. 756, the crura of the stapes were removed and the footplate studied under the microscope, revealing diffuse red vascular otosclerosis with complete marginal footplate fixation.

IMMEDIATE RESULTS.

The immediate results of this more complex stapes surgery is based upon the 30-day audiogram. The statistical results reported are not intended to be final results. The statistics are reported on the Otosclerosis Study Group form, as sug-

CHART No. I.

CRITERIA FOR REPORTING DEAFNESS IN THE SURGERY OF CONDUCTIVE DEAFNESS.

1. Date of Report: January 20, 1959.
2. Period of Time during which surgery being reported was performed: May 20, 1958 to Dec. 10, 1958.
3. Number of Cases operated during this period: 100; 19 Bi-crural otosclerosis.
4. Number of Cases available from 3 with one month of later tests for this report: 100 per cent.

HEARING GAIN.

Class	No. Cases	Pathology	Operation	11 db or more No.	%	Hearing Level Over 30 No.	%	30-40 No.	%	No Gain No.	%	Losses No.	%
A	7	BiC	4+ CR	6	86	4	57	2	29	1	14		
B	7	BiC	4+ CR	6	86	5	71	1	14			1	–12
C	2	BiC	4+ CR	2	100			1	50				
D	3	BiC	4+ CR	2	66			1	33			1	33

PER CENT OF IMPROVEMENT.

Class	No. Cases	– % No.	%	0-24% No.	%	25-49% No.	%	50-74% No.	%	75-99% No.	%	100% + No.	%
A	7			1	14	3	43	1	14	1	14	1	14
B	7	1	14					1	14	4	57	1	14
C	2							1	50	1	50		
D	3	1	33							2	66		

Chart No. 1—The Audio-analysis of Cases of Crural Repositioning for BiCrural Otosclerosis is reported on a form suggested by the Otosclerosis Study Group, with addition of a column for the classification of the type of pathology and the type of Stapes Operation (BiC = BiCrural; CR = Crural Repositioning.) The hearing gain is also subdivided.

gested by Dr. Eugene Derlacki and his colleagues on this committee (Chart Nos. 1-a, b). Because of our interest in the surgical pathology of the stapes, the relationship of the surgical pathology to the slope of the audiogram, and in computing the results of the various types of operations, I would advise including these categories immediately after the classifications into Groups A, B, C and D. I would also add the 90-day, 180-day and 360-day findings, as suggested in the IBM form of Dr. John Shea.

CLASSIFICATION A, B, C, D.

A review of the preoperative bone conduction audiogram shows that 37 per cent are in Category A, 37 per cent in Category B, 10 per cent in Category C, and 16 per cent in Category D.

Of the A-cases, 86 per cent showed a gain of 11 db or more. Of the 7 A-cases four, or 57 per cent, were at the 30 db level or better, an average hearing level of 24 db. Two cases were between the 30 db and 40 db level, and had an average hearing of 33 db. One case showed no gain in hearing and has had a successful fenestration. No case showed a hearing loss. The air-bone gap was closed 75 per cent or more in two cases; 50 to 74 per cent in one case; 25 to 49 per cent in three cases; less than 24 per cent in zero cases; and no gain in one case.

Of the B-cases, 86 per cent showed a gain of 11 db or more. Of the seven B-cases five, or 71 per cent, were at the 30 db level or better, an average hearing level of 19 db. One case was between the 30 and 40 db level and had an average hearing of 33 db. One case showed a hearing loss of 12 db. The air-bone gap was closed 75 per cent or more in five cases; 50 to 74 per cent in one case; 25 to 49 per cent in none; less than 24 per cent in none, and no gain in one case.

Of the C-cases, 100 per cent showed a gain of 11 db or more. Of the two C-cases, none attained the 30 db level or better. One case was between the 30 and 40 db level, and had a gain of 25 db to the 37 db level. One case had hearing at the 48 db level but a gain of 30 db, and improvement in ability

to lip read and use a hearing aid. The air-bone gap was closed 75 per cent or more in one case and between 50 and 74 per cent in the other.

Of the D-cases, 66 per cent showed a gain of 11 db. Of the three D-cases, none attained the 30 db level or better. One D-case attained an average hearing level of 35 db, with a gain of 37 db. One recent patient experienced a 41 db gain to the 43 db level, but this has regressed to a 20 db gain, either from re-fixation or loss of ossicular continuity. One case showed a hearing loss of 10 db. The air-bone gap was closed 75 per cent or more in the other two cases.

LABYRINTHINE DISTURBANCE.

With this much stapes surgery, I was concerned about persistent postoperative vertigo. In response to the questionnaires, two patients stated that they have slight dizziness.

LONG TERM RESULTS.

The long term audiograms are not yet available on these cases performed in mid-1958 after crural repositioning became a standardized procedure for bi-crural otosclerosis. We do have isolated audiograms on individual cases showing phenominal hearing gains and hearing levels retained for more than one year, with the longest case 20 months postoperative.

It is my hope that long-term audiograms will be available before publication. Current response to the questionnaire indicates that of those who experienced hearing improvement, four patients have lost their hearing gain, leaving well over 50 per cent who have retained their hearing improvement and classify their hearing as good or very good.

SUMMARY.

1. Otosclerotic involvement of both stapedial crura is pathologically beyond the limits of reasonably predictable total stapes mobilization surgery.

2. Bi-crural otosclerosis is a positive indication for specialized surgical techniques.

3. When there is bi-crural otosclerosis and a reasonable area of normal or near-normal stapedial footplate, I advocate central mobilization of the uninvolved footplate and central repositioning of the posterior crus as a possible method of maintaining hearing improvement.

4. Central repositioning of the posterior crus is accompanied by tendonotomy, anterior crurotomy, and central footplate mobilization. The technique of central repositioning is described.

5. Artificial cruralization is indicated in bi-crural otosclerosis when the crura are extensively involved and there is a

CHART NO. II.—STAPES OPERABILITY.

SURGICAL PATHOLOGY.	APPROPRIATE OPERATION.
1. Anterior Footplate Otosclerosis; Superficial footplate.	Direct mobilization, pick inserted anteriorly.
2. Anterior Footplate; Deep Footplate.	Indirect mobilization at incus.
3. Anterior Crural Otosclerosis.	Anterior crurotomy; mobilization posterior footplate and crus.
4. Anterior Crural, Superior or Inferior Marginal Otosclerosis.	Anterior crurotomy; mobilization posterior footplate and crus.
5. Bi-Crural Otosclerosis; Some normal footplate, wide niche.	*a.* Anterior crurotomy and mobilization central footplate and crural repositioning.
Same, narrow niche.	*b.* Mobilization central footplate and artificial cruralization.
6. Complete Footplate Otosclerosis; Thin (Annulus visible, Superficial Footplate).	Circumferential footplate mobilization.
7. Complete Footplate Otosclerosis, Thick (Annulus not visible).	Oval window graft; Lempert fenestration.
8. Obliteration of oval window niche.	Lempert fenestration usually required, but if there is a small area of deep, normal blue footplate, de-crural footplate mobilization or artificial cruralization may be attempted.
9. Revisions of the stapes mobilization is indicated only if the primary pathology was favorable and the primary surgery inadequate, or inappropriate for the specific pathology.	
10. Revisions of the stapes mobilization are not indicated in the presence of primarily unfavorable anatomy and pathology; these are indications for the fenestration operation.	

Chart No. 2—Surgical judgment is required to evaluate the surgical pathology of the stapes in order to apply the appropriate surgical technique.

reasonable area of normal or near-normal footplate, or when there is marked narrowing of the deep, oval window niche, artificial crura need not be employed indiscriminately.

6. Footplate windows and oval window grafts may be indicated in complete footplate otosclerosis in cases not suitable for the fenestration operation.

7. In complete footplate otosclerosis, with an adequate air bone gap, the modified Lempert fenestration operation is my procedure of choice.

8. At the present time, most all stapes surgeons are developing their own techniques for overcoming the more complex varieties of stapes pathology. It is my sincere hope that this posterior crural repositioning will be of lasting benefit in some cases of bi-crural otosclerosis.

9. With the operating microscope and modern surgical instruments, it is possible to do almost anything to the stapes. Surgical judgment is required to do the right thing. My current opinion as to pathological indications for the various types of stapes operations in indicated on Chart 2, showing the basic classification of stapes pathology, and the corresponding indicated basic types of stapes operations.

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ULTRASONIC THERAPY FOR RELIEF OF VERTIGO DUE TO MENIERE'S DISEASE.*†

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Since Langevin in 1916 started studying the effects of ultrasound there has been a steadily increasing interest, so that today to glance through the literature is to be astounded at the volume of publications.

If we confine our attention to those reports of interest to the otologist we find that some few used ultrasound in the treatment of otosclerosis in the years 1928-1933.[1] Due to the equivocal results achieved nothing more appeared until 1949[2]; however, since then there has been increased interest, and the work of Krejci[3] stimulated others to apply ultrasound to the vestibule in experimental animals and directly to the horizontal canal in the treatment of Meniere's disease[3,4] Today there are a number of reports on the clinical use of the beam in this condition.

Undoubtedly the experience of Arslan[5] is the most extensive but reports of Portmann, et al.,[6] Dubs[7] and Lumsden[8] are worthy of study.

METHODS OF PRODUCING ULTRASOUND.

Ultrasound is sound which has a frequency above 18 kilocycles or above that which can be detected by the human ear. There are three ways in which ultrasound can be produced: *mechanical, magneto striction,* and through *piezoelectric* effect. This last is the method used clinically as only with it can radiation of sufficient frequency and intensity be obtained.

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When electrical charges are applied to the surfaces of a crystal, vibrations of the crystal result. One of the most suitable crystals is quartz and is most frequently used.

PHYSICS.

Generally, ultrasound behaves in the same fashion as audible sound, but there are one or two differences which are of clinical importance. Vibrations travel four times faster in liquids and 20 times faster in solids than in air.

Though sound is transmitted with ease and uniformity through homogeneous media, at the interfaces of two dissimilar conductors, such as quartz and air, or even water and air a large portion of energy is reflected; furthermore, at such interfaces there develop waves traveling at right angles to the axis of the beam.

METHODS OF APPLICATION TO EAR.

1. *External mastoid.* Here the beam is applied to the skin overlying the mastoid process.

2. *Transmeatal.* This is the method as described by Portmann and Barbe.[9]

3. *Direct application to the bony horizontal canal.* This is the method as used by Krejci, Arslan, Lumsden and Dubs.

From the preceding section on the physics of ultrasound it would appear that the last method, with direct application to the horizontal canal would be most likely to produce an effect localized to the vestibular receptors. When using the first and second methods for treatment of the vestibular receptors there would be considerable loss of energy at the interfaces, and the beam could not be directed accurately.

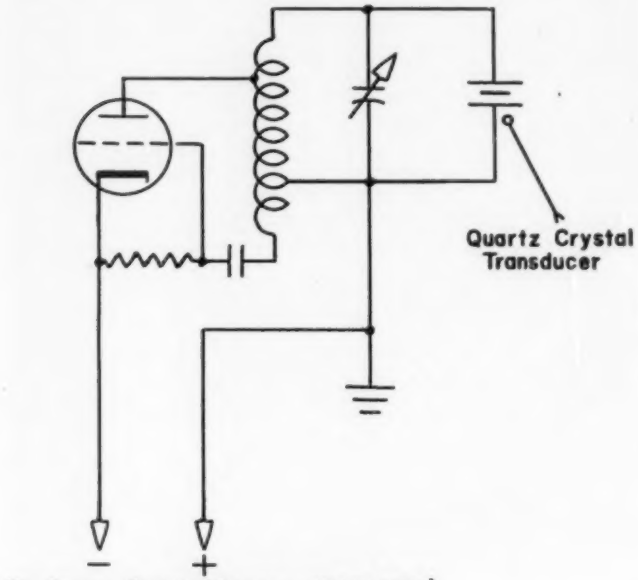
EQUIPMENT USED.

The machine used is that developed by an Italian company for Professor Arslan. It consists essentially of a high frequency generator connected to the erogating head which is water cooled (see Figs. 1, 2, 3, 4).

Fig. 1. Photograph of the Ultrasonic Apparatus.

The area of the circular flat tip of the erogating head is 0.2 sq. cm., and the frequency is approximately 950 kilocycles per second as tested by the ultrasonic probe. The intensity dial on the machine is supposed to give the intensity of the delivered beam and not the intensity output of the electrical circuit; furthermore, there is a frequency control dial, but this is only effective over a limited band of approximately 40

cps and is used as a fine control to set the beam at the start of the procedure, and during the operation also to offset changes in the frequency resulting from changes in temperature, amount of fluid or blood at the tip and also changes in the position of contact between tip and bone of horizontal canal.



SIMPLIFIED SCHEMATIC DIAGRAM
OF ULTRASOUND GENERATOR

Fig. 2.

BIOLOGICAL EFFECT OF ULTRASOUND.

It is known that ultrasound has three effects: *1.* Thermal; *2.* mechanical; *3.* chemical. That they are of varying importance in different fields is known, but their relative importance in the ear has not yet been shown by experimental work so far undertaken. That heat to a considerable degree

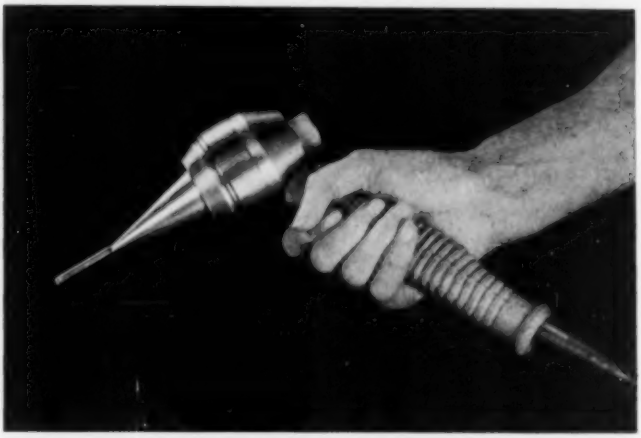


Fig. 3. Photograph of the Erogating Head.

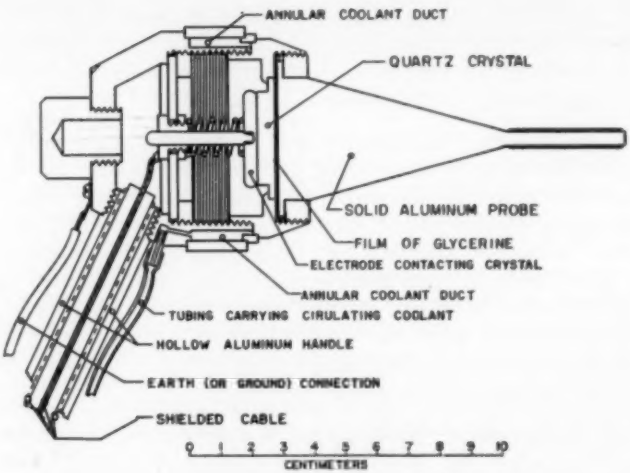


Fig. 4.

is produced by ultrasound applied to the ear has been shown, but whether this is the main effect has not been proved. Histological changes produced by ultrasound have been studied by Krejci and Bornschein,[3] Portmann, Portmann and Barbe,[9] Naumann[10] and de Stefani.[11] Degrees of effect from hyperemia to tissue necrosis have been observed, and this depends on the dosage and technique used. Of great interest is the work of de Stefani which shows the destruction of the neuroepithelium with preservation of the cupula.

TECHNIQUE.

The method used is similar to that described by Professor Arslan.

The average patient receives sodium pentobarbital 180 mg. two hours and Meperidine hydrochloride 100 mgm. 30 minutes before operation as premedication. Local anesthesia is then induced and a cortical mastoidectomy performed. It is essential to gain a good exposure of the horizontal canal to permit correct positioning of the erogating head. Four of the operations have been performed through the postaural route (WMSI) and two by the endaural (JRL) and there seems to be no justification for the claim that the postaural route is preferable to gain the necessary exposure.

Gaining through the experience of Arslan, we apply the tip towards the posterior end rather than on the convexity of the horizontal canal. In this manner we have escaped the unfortunate early experience of Arslan and Lumsden with facial palsies. Prior to applying the erogating tip the posterior part is brought into prominence and the surface flattened by careful removal of bone. This is done to insure good contact, the importance of which has already been indicated. Thorough hemostasis is then secured, and a fine suction tip placed in the posterior part of the cavity.

During the period of erogation a second assistant is required to operate the machine and watch the eyes for nystagmus and the face to detect any twitching indicative of facial nerve stimulation.

The dosage applied varies from patient to patient, but

fortunately the observation of induced nystagmus gives a reliable guide as to dosage required. Due to failures in the apparatus the duration of application and the intensities used have varied considerably. The duration varied from 70 minutes to 15 minutes. The intensity has also varied considerably from starting at 6 watts per sq. cm. up to 12 watts per sq. cm. With the recent acquisition of an ultrasound probe we are now in a position to determine more accurately the frequency and intensity of the beam and detect early any defect in the machine.

In the last three cases the starting intensity has been 8 watts per sq. cm. After one to two minutes "irritative" nystagmus or nystagmus to the same side appears. This is usually brisk and is sustained. At this point the intensity is increased to 9 or 10 watts per sq. cm. This irritative nystagmus continues for a varying period when "paralytic" nystagmus, or nystagmus to the opposite side, ensues. There may be an abrupt change from one type to the other, or there may be a period of a few minutes when there are brief episodes of the paralytic form reverting to irritative until finally the paralytic nystagmus becomes constant, or there may be a few minutes between the cessation of irritative nystagmus and the onset of paralytic nystagmus when there is no defiinite movement of the eyes to one or other side.

Occasionally a rotatory component is noted and this results, we suspect, from the beam affecting the posterior end of the posterior vertical canal. As indicated, contact between erogating tip and bony horizontal canal is made towards the posterior part of the canal. This has been done from the outset as it was Arslan's experience that facial paralysis, even though of only a temporary nature, can ensue if the beam is directed too far anteriorly. One should also avoid directing the beam towards the cochlea or disturbing the incus. It is known also that the desired clinical effect on the neuroepithelium placed anteriorly can be obtained even when erogating posteriorly.[12]

The erogating head is removed when the paralytic nystagmus has been constant for one to four minutes. At those times when the machine was obviously not functioning properly and periods of up to 66 minutes were required to induce

paralytic nystagmus, the tip was left in position for an additional four minutes. When the paralytic nystagmus appeared in 14 minutes then only one minute of further treatment was given. Gelfoam soaked in penicillin was then placed in the cavity and the incision closed completely without drainage.

Even the extremely nervous patient has withstood the procedure well in spite of the intense vertigo experienced. In only one case has vomiting occurred.

POSTOPERATIVELY.

The vestibular disturbance induced by the procedure is quite marked and spontaneous nystagmus of paralytic nature,

TABLE I.

Results Obtained by Ultrasonic Therapy.

Number	Application of US	Vertigo	Tinnitus	Hearing
1	65 minutes	Improved	Never present	No change
2	70 minutes	Free	Reduced	Improved
3	30 minutes	Improved	Unchanged	Worse
4	31 minutes	Free	Reduced	Improved
5	60 minutes	Free	Reduced	Worse
6	15 minutes	Free	Reduced	Improved

that is to say to the opposite side, has been noted up to the sixth day; however, usually within 48 hours this spontaneous nystagmus has cleared, and the patient can be up but is still experiencing vertigo on any movement of the head. The patient is usually fit to return home at the end of eight days. Generally it has taken the successfully erogated patients up to two and a half or three months to be free of the results of the operation—one would expect such a period of time to elapse before compensation developed.

RESULTS.

It is well to state at this stage that the six patients subjected to this procedure had been exposed to the usual conserative therapeutic measures for periods from 17 months to three years prior to operation, and none of them had gained any appreciable benefit. The youngest was 31 years and the oldest 57 years.

VERTIGO.

Four have gained complete freedom from vertiginous attacks and two have improved. One of the latter unfortunately is showing signs and symptoms of involvement of the other side. Arslan reports success in 94 per cent of 195 cases and Lumsden 19 (four with modified attacks) out of 22.[9]

Following the caloric responses pre- and postoperatively has been of considerable interest. Two patients were subjected preoperatively to the modified Kobrak test and showed diminished response on the affected side. Four were tested

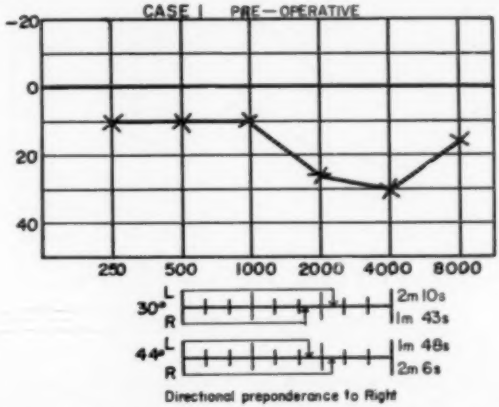


Fig. 5. Case 1 illustrates one preoperative audiogram in the affected ear, also, the caloric responses on that occasion. The audiogram on this date was not characteristic of Meniere's disease.

by the differential caloric test and the results were, two canal pareses, one preponderance to the opposite side and one combined lesion of the same side.

Immediately following removal of the erogating tip from the cavity in two patients, warm water at approximately 45° C. was run into the cavity. In both cases this induced an inhibition of the paralytic nystagmus to the opposite side, whereas when the sucker was applied just over the horizontal canal causing cooling, the paralytic nystagmus was enhanced. By

the end of two weeks, when the ear has healed, there is generally no response to water at 18° C. though it may take up to three months or longer for disappearance of the response to 10 cc. water at 4° C.

This amply confirms what has been shown in animal experiments that the lesion caused by ultrasound is not complete immediately but is progressive over a period of weeks or even months; further, the lesion may be of a temporary nature.[2] This is shown in Case 1. Preoperatively (see Fig. 5) the

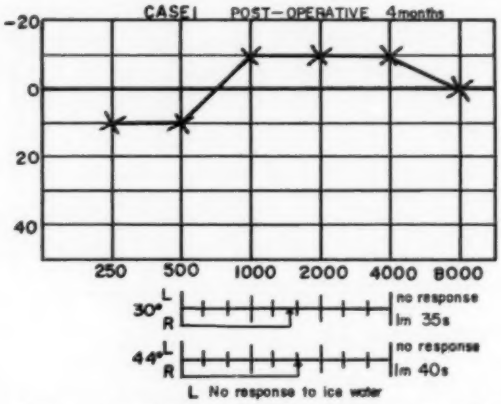


Fig. 6. Case 1. AC audiogram four months after ultrasonic treatment. Note absence of caloric response in left ear and lowering of the AC threshold.

differential caloric had a pattern of directional preponderance to the opposite side. Four months postoperatively (see Fig. 6) there was no response to 30° C. water during the differential caloric, and 10 cc. of 4° C. water failed to elicit a response; however, 15 months postoperatively (see Fig. 7) the differential caloric responses are within normal limits. The patient has had, however, definite benefit from the operation. For 17 months prior to operation this patient had been under conservative therapy without benefit. For two months prior to operation she had been confined to the house, had to have help to care for her children and had withdrawn socially.

Within two and a half months of the operation she had renewed her social activities and again enjoyed her children. She stated recently (three weeks ago) that though she occasionally feels unsteady she has had no true vertiginous attacks as experienced prior to the operation.

In two patients during the period after the loss of response to water at 18° and while 4° C. water still stimulates, we have observed induced nystagmus to the same side, the reverse of what one would expect.

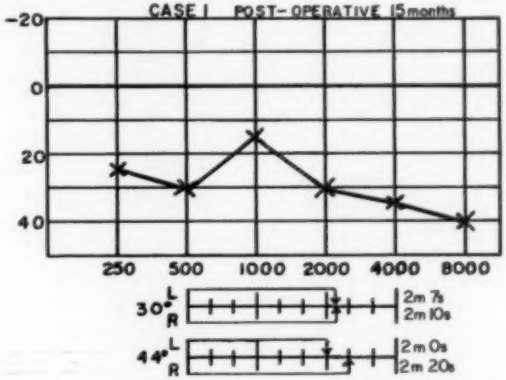


Fig. 7. Case 1. AC threshold is again elevated. Caloric responses are within normal limits at 15 months.

Although the patient can get about without too much difficulty within ten days after operation, quick head movements may cause discomfort up to two or three months; also positional nystagmus has been observed up to two months. This we take to indicate that the ultrasound induces only a partial destruction of the vestibular system.[13]

TINNITUS.

Five of the six patients experienced tinnitus prior to operation. Of these five, one has sensed no change, while in four the intensity has been reduced, and where intermittent, the frequency reduced, following the operation.

HEARING.

Arslan[5] has reported 40 to 50 per cent of cases of Meniere's disease gaining an improvement in hearing following the application of ultrasound, but Lumsden found that only one out of 22 cases showed improvement. Our cases have not been followed for a long enough period to assess the effect of ultrasound. That complete loss of hearing has not resulted is at least certain. The most recent audiograms indicate that some improvement has occurred in three cases (see Figs. 8, 9),

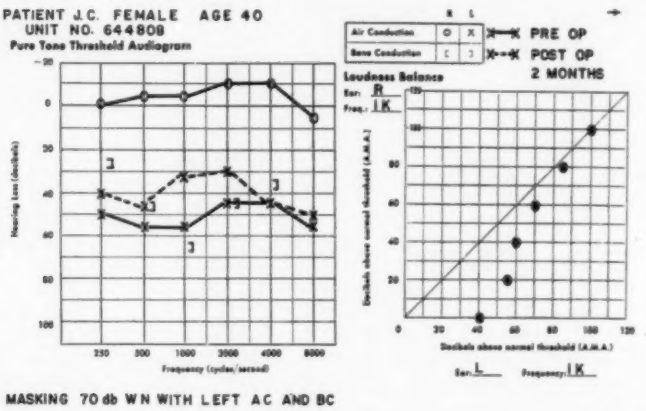


Fig. 8. Case 4. Typical audiogram and loudness balance test for Meniere's disease, left ear. Note apparent improvement in hearing two months after treatment.

one remains unchanged and two have lost some hearing since the operation.

As seen in Case 1 there are considerable changes in the threshold during the postoperative period (see Figs. 6, 7), as well as in the preoperative period, so that unless one is in the position to compare a series of preoperative audiograms taken during and between attacks with a series of audiograms over many months or even a year in the postoperative period, it is impossible to assess accurately the effect of ultrasound on the hearing; furthermore, if one takes the variations in

the threshold as an indication of the continuation of the disease process albeit without vertiginous attacks, and there are symptoms and signs to suggest this, then it would be surprising to achieve a true and permanent lowering of the threshold.

COMPLICATIONS.

In two cases the middle ear contained blood for approxi-

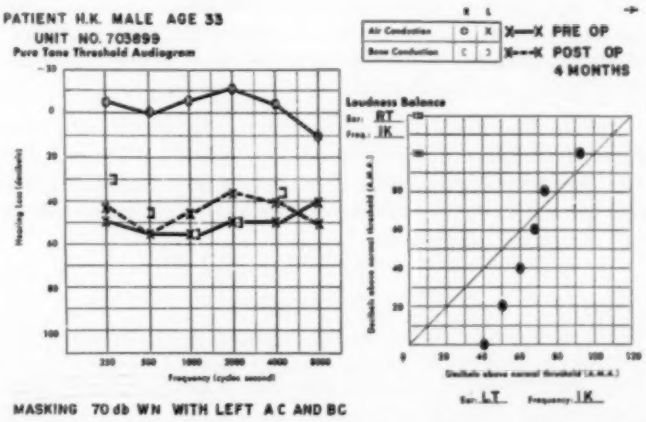


Fig. 9. Case 6. Meniere's disease, left ear. Note slight apparent improvement in hearing four months after operation. Such shifts in the threshold are characteristic of Meniere's disease and may not indicate improvement due to the treatment.

mately two weeks after the operation, but this cleared spontaneously.

In one a wound abscess developed necessitating re-hospitalization and antibiotic therapy.

No facial paralysis even of a temporary nature was encountered.

CONCLUSION.

Although the dosage used in this series of cases has been

variable and in some inadvertently conservative, the results obtained in the control of attacks of vertigo have been consistently favorable.

Hearing has been retained in all cases.

There has been no case of facial palsy. Recent reports by Arslan and Lumsden in larger series of cases also give reassurance that permanent facial nerve injury may be avoided providing that adequate care is used during treatment.

In view of the experience with this small group of cases, supported by the experiences of others, we feel justified in using ultrasonic therapy in preference to other methods of labyrinthine destruction.

Certain findings in the postoperative audiograms and caloric responses are presented without explanation. They indicate a need for further study of the peripheral vestibular system. Also the effect of ultrasound on the receptor organs requires more precise investigation.

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UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The Department of Otolaryngology, University of Illinois College of Medicine, announces two special postgraduate courses to be offered in the Fall of 1959.

ANNUAL OTOLARYNGOLOGIC ASSEMBLY.

The Assembly will be conducted September 18-26, 1959, and will consist of a series of lectures and panels concerning advancements in otolaryngology. Some of the sessions will be devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat. Guest lecturers will participate in an entire day's program reviewing the latest advances and principles of temporal bone surgery.

Chairmen of the Assembly are Maurice F. Snitman, M.D., and Emanuel M. Skolnik, M.D.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The course in laryngology and bronchoesophagology, under the chairmanship of Paul H. Holinger, M.D., is scheduled November 9-21, 1959.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

EVALUATION OF LARYNGECTOMY WITH RADICAL NECK DISSECTION.*

JOHN J. O'KEEFE, M.D.,

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Carcinoma of the larynx of extra-cordal origin, or of cordal origin but with gross extension to structures adjacent to the cord, are more malignant lesions than are those cancers confined to the true vocal cords. This is attested to in the high incidence of metastases associated with them. Not only are such lesions frequently of a higher anaplastic histologic classification, but anatomically they possess the potential of metastasizing relatively easily. Additionally, the lesions of extra-cordal origin are usually asymptomatic in their early growth, thereby allowing for lymphatic invasion before arousing subjective complaints.

The detectable presence of cervical lymph nodes, in proven instances of laryngeal cancer, is presumptive evidence of regional metastases. When such nodes are ascertained preoperatively, there is no question that they constitute a positive indication for radical neck dissection as a component part of the primary operative procedure, regardless of the location, extent, or stage of development of the laryngeal growth; however, in instances of similarly classified laryngeal lesions, but which are further characterized by impalpable cervical lymph nodes, a real and serious controversy exists disputing the necessity for such extensive or additional surgery.

In appraising this issue, it is of critical importance to bear in mind that inability to palpate cervical lymph nodes preoperatively does not constitute the criterion by which one adjudges the rationale of combining neck dissection with laryngectomy for the resection of such laryngeal lesions; rather, it is because of the known behavior pattern of these

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cancers, that their location, either by origin or by extent, is incriminated as the principal characteristic by which their ability to metastasize is determined. This, therefore, is the criterion which would influence and direct such judgment. It is equally important to realize that cervical metastases, either gross or microscopic, exert a grave influence on mortality. The greater seriousness of this problem lies in the fact that the disease has now extended beyond the confines of the larynx, and threatens general body dissemination. At best, prognosis is guarded, for, as with the evaluation of cancer generally, prognosis is considered favorable only when the primary lesion is small, when it is situated wherein lymphatic drainage is scant, and when regional metastases cannot be demonstrated.

CLASSIFICATION OF MATERIAL.

Even under optimum circumstances, an accurate classification of a laryngeal tumor is often a difficult task. This is especially true in the present study, wherein the tumors under consideration are essentially far advanced and extensive ones, with many of their characteristics distorted or hidden. Tumors whose site of origin cannot be discerned, are classified according to the structure involved by the greatest amount of growth, even though more than one anatomic part may be involved. Palpable cervical lymph nodes are considered metastatic lesions, and are evaluated preoperatively only as resectable or not.

Laryngectomy with unilateral neck dissection, as a one-stage procedure, was performed in 153 cases. This operative method is referred to as prophylactic or protective when neck nodes are impalpable, and as definitive when neck nodes are palpable; accordingly 85 of these operations were definitive procedures, and 68 were prophylactic. Laryngectomy alone, since 1950, was performed in only 107 cases. Failures relative to this operative method, manifested as metastases to the regional lymph nodes, were operated by secondary radical neck dissection. The results obtained by these various operative procedures will be evaluated and correlated with the criteria by which they were selected as the operation of choice.

I. DEFINITIVE LARYNGECTOMY WITH RADICAL NECK DISSECTION.

Palpable cervical lymph nodes, associated with a proven carcinoma of the larynx, constitute a positive indication for resection by laryngectomy with neck dissection. In arriving at this decision, one should not be influenced by the size or location of the laryngeal lesion, nor by the size or number of palpable cervical nodes. Any other method, or any method of less extent, is contrary to the accepted principles of cancer surgery.

The following statistics are practical applications of this argument. In the series of 85 definitive operations, the laryngeal tumor in 37 instances was located supraglotically; in 31 instances it was cordal, but with gross extension either anteriorly or posteriorly; and in 17 instances its principal

TABLE I.

Laryngectomy with Radical Neck Dissection—Cervical Nodes Palpable.

Location	Number of Cases	Nodes Positive for Cancer	Per Cent
Supraglottic	37	24	64.8
Cordal	31	18	58.0
Subglottic	17	10	58.8

location was subglottic. In the supraglottic group, the associated cervical lymph nodes contained cancer in 24 cases, 64.8 per cent; in the extensive cordal group, lymph nodes were positive for cancer in 18 cases, 58 per cent; and in the subglottic group, these nodes were positive in 10 cases, 58.8 per cent.

The proximate similarity of these percentage rates, reflecting the incident presence of cancer in the palpable cervical lymph nodes in these three groups of patients, factually supports the premises that laryngeal cancer, either originating or extending beyond the confines of the true vocal cord, is readily capable of metastasizing, and that associated palpable cervical lymph nodes should be presumed to represent malignant metastases. Conclusive evidence of the malignant potential of these lesions is revealed in the fact that in spite

of such extensive sugery, 20 of these patients are dead of cancer, 23.5 per cent.

II. PROPHYLACTIC LARYNGECTOMY WITH RADICAL NECK DISSECTION.

Laryngeal carcinomas, as described and classified previously, but which are further characterized by impalpable cervical lymph nodes, are said to constitute a relative indication for resection by laryngectomy with radical neck dissection. "Relative" is interpreted to mean that the operator has a choice in his selection of operation, which choice assumedly derives from the absence of palpable cervical lymph nodes. This is an unwarranted and unfounded reason for proposing a choice of operation. Having established the significance of location, as the principal if not the only characteristic which determines

TABLE II.

Laryngectomy with Radical Neck Dissection—Cervical Nodes Impalpable.

Location	Number of Cases	Nodes Positive for Cancer	Per Cent
Supraglottic	28	9	32.1
Cordal	18	2	11.1
Subglottic	22	5	22.7

the ability of these laryngeal tumors to metastasize, this characteristic should be the sole determining factor in evaluating the need for adding neck dissection to laryngectomy for their resection. The inability to palpate regional lymph nodes is irrelevant to the issue.

The second part of this study is presented in support of this assertion. Sixty-eight prophylactic combined operations were performed on patients classified as noted above, and who in common were adjudged to have no evidence of regional metastases preoperatively. The laryngeal tumor was located supraglottically in 28 instances; grossly cordal in 18 instances, and in 22 instances its principal mass was subglottic. The lymph nodes contained in the resected cervical tissues were histologically positive for cancer in 16 cases, 23.5 per cent; specifically in nine of the supraglottic group; in two of the cordal cases, and in five of the subglottic group.

These 16 cases surely would have developed overt metastic lesions had the primary surgery been limited to laryngectomy alone. Undetected or undetectable metastic cancer occurring in approximately 25 per cent of patients is reason enough to consider this type of laryngeal lesion an absolute indication for resection by laryngectomy with radical neck dissection. Again, it is stressed that inability to palpate cervical lymph nodes is not relevant to this issue. Of utmost importance is the fact that 12 of these 16 patients are alive and apparently free of cancer for more than three years postoperatively. The four deaths, 5.8 per cent, are all attributable to metastic disease.

III. PRIMARY LARYNGECTOMY.

Between 1950 and 1955, only 107 cases of cordal cancer were operated by total laryngectomy. This relatively small number

TABLE III.

PRIMARY TOTAL LARYNGECTOMY.

Number of Patients Operated	107
Number of Failures	31
Per Cent of Failures	28.9

is a consequence of the increasing number of combined operations being performed as primary surgical procedures. The laryngeal lesions, although classified cordal in location, evidenced gross extension anteriorly or posteriorly, or both, so that commonly there was involvement of the anterior commissure or of the opposite cord, and/or fixation of the arytenoid. Essentially, they constitute extensive cordal cancers, without demonstrable signs of palpable cervical nodes.

Laryngectomy failures, noted as malignant metastases to regional lymph nodes, occurred in 31 instances, 28.9 per cent. Twenty-five of these were operated by secondary radical neck dissection, and two were irradiated. One of the irradiated patients is alive and free of disease more than five years after therapy; the other is dead of cancer. Sixteen of the 25 operated patients are alive and well, three or more years postoperativeiy; a proximate salvage rate of 64 per cent,

but a total salvage rate of only 51.6 per cent. Ten of the 31 primary failures are dead of cancer, 32.2 per cent.

This group of patients, representative of extensive cordal cancer without regional metastasis, presumably had been properly selected for primary laryngectomy; yet the initial failure rate of almost 30 per cent contradicts the assumption that cordal cancers are not prone to metastasize, and at the same time, signifies the inadequacy of this operative method.

DISCUSSION.

Cancers of the larynx classified as extra-cordal, whether by origin or by extent, are potentially highly malignant. Their degree of malignancy derives from their location rather than from any other characteristic, and is objectively assessed through the identification of regional metastases.

The successful surgical treatment of these forms of laryngeal cancer is inherently dependent upon an acceptance of this appraisal. This is borne out in the present study where, in employing total laryngectomy with radical neck dissection as the only operative procedure, the failure rate incident to the resection of laryngeal lesions associated with palpable cervical lymph nodes preoperatively, was four times greater than that resulting from operating the same type of laryngeal lesions, but which were free of demonstrable preoperative cervical metastases. The resected cervical nodes, in 85 patients operated by definitive laryngectomy with neck dissection, were positive for cancer in approximately 60 per cent of cases; the nodes contained in the tissues resected through operating 68 patients by prophylactic laryngectomy with neck dissection, were positive for cancer in 23.5 per cent. This, in summary, is the crux of the problem. Cervical metastases occur easily and relatively early in laryngeal lesions of extra-cordal location; that for the successful resection of these cancers, one should anticipate the development of such metastases and make adequate provision for their resection; and that "prophylactic" is actually a superfluous term in describing the appropriate method of resection.

Additionally and somewhat parenthetically, the malignant

potential of diffuse cordal cancers is little less than that of cancers of extra-cordal location. The 29 per cent laryngectomy failure rate of this carefully classified group of patients, further supports the rationale of recommending more extensive surgical measures as primary resection techniques, even for lesions of this limited extent. In so doing, the basic tenet of cancer surgery, that of saving life, will be better served.

TEMPLE UNIVERSITY SCHOOL OF MEDICINE AND HOSPITAL.

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TRIGEMINAL NEURALGIA—TIC DOULOUREUX.*

JAMES P. RIGG, M.D.,
and
ROBERT W. RIGG, M.D.,
(By Invitation),
Grand Junction, Colo.

Recently Dr. Stanley P. Reimann, of Philadelphia, stated: "Tomorrow is being born today in medical research laboratories. We have no crystal ball, and it may be folly to prophesy what will be discovered, but much progress is evidenced everywhere . . . As ever, more accurate instruments are being given to us by our friends in mathematics, physics, engineering, and in many other fields, thereby presenting many new facets of investigation.

"The ideal of the doctor is that no one should die of disease, but should live his allotted life span and then quickly fall apart like the famous one-horse shay. We are far from this ideal, but it is a goal we are all striving for."

Pain is an ever-present problem in medicine, and we, as doctors, are striving constantly to alleviate this. It is our hope and our desire that pain of any type can be adequately controlled, and permanently dissipated. Concurring with Dr. Walter Scott that "man can look forward with eager anticipation to continuing progress in the medical sciences, but the unfolding of each new step will likely be evolutionary, rather than revolutionary. Research is currently knocking at the door which will soon open and lead to worldwide control of viral diseases."

Dr. Scott further states: "Degenerative processes, particularly arteriosclerosis, together with the ever-increasing turmoil in man's emotional life, will be conquered more slowly and along lines not presently known; however, I do not expect

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that our descendants, a thousand generations hence, will ever see eradication of all congenital deformities or all allergies."

By the same token, we can only hope there will be definite progress in the field of trigeminal neuralgia, and that the cause back of this lancinating, excruciating, agonizing, horrible, unbearable pain will be found. Trigeminal neuralgia is primarily an affliction of older persons, although it may occur at any age. It must be remembered, that as we have more elderly people and especially elderly women, we will be confronted oftener with this painful condition causing lancinating, paroxysmal pain in one or more branches of the Vth cranial nerve. This is, indeed, a new frontier to conquer.

Only certain specialized nerve endings are capable of receiving pain stimuli. The trigeminal nerve is composed of both sensory and motor fibers; the motor fibers are distributed to the muscles of mastication. The sensory fibers through the three branches of the nerve are distributed over the forehead, cheek, nose and mandible, and in front of and superior to the ear.

The sensory nuclei of the trigeminal nerve extends over the entire length of the mesencephalon and rhomencephalon. The main sensory nucleus, transmitting touch sensations, is in the region of the pons. The muscles of mastication are the temporals, masseters and pterygoids.

It has been stated that the occurrence of trigeminal neuralgia, as an early symptom associated with multiple sclerosis, has long been noted. The simultaneous unrelated occurrence of these two conditions in the same individual seems worth investigating.

Motor root supplies the masseter, temporal, internal and external pterygoids and the tensor tympani, tensor palati, mylohyoid and anterior belly of the digastric. The second branch of the trigeminal, from the sensory standpoint, sends root fibers into the pons, which descend in the medulla to the second cervical segment of the spinal cord, the ophthalmic division descending farthest. The Gasserian ganglion, which lies on the petrous portion of the temporal bone in the middle cranial fossa, divides into an ophthalmic, maxillary and mandi-

bular division. The lingual branch of the mandibular carries the taste fibers of the chorda tympani. The ophthalmic nerve gives off sympathetic fibers to the lacrimal gland. The trigeminal stands in relation to the VIIth nerve by means of the sphenopalatine ganglion, and the glossopharyngeal by way of the otic; it is also connected with the facial by means of the large and small superficial petrosal nerves. Besides supplying sensory fibers to the face, temple, forehead and anterior part of the scalp, it also innervates the external ear in part (not the whole internal meatus, which is supplied partly by the facial, and the vagus), and it supplies the cornea (pain and not touch fibers), conjunctiva, lingual, gingival, buccal, upper pharyngeal and nasal mucous membrane, the middle ear and the dura. Deep facial sensation runs in the facial nerve.

OCCURRENCE.

Harris reports a patient on whom he did a Gasserian ganglion injection at the age of four-and-a-half years. She had been suffering since the age of 16 months from frequently repeated daily spasms of pain, lasting a few seconds only, and referred to the left lower jaw up to the ear.

There have been a few cases reported, prior to and in the teenage period, but this is extremely rare. The upper limit of age, of course, is even toward nonegenarians. Most cases, however, are elderly, edentulous females. The ratio of females over males is approximately three or four to one. Two per cent of cases are bilateral. Trigeminal neuralgia may occur more specifically with glossopharyngeal neuralgia concomitantly and with other facial neuralgias.

ETIOLOGY AND PATHOLOGY.

1. Neurovascular.

2. Traction (dura), (constricting dural bands along the bony petrous ridge).

3. Emotional—psychic.

4. Infectious, sinusitis, dental, systemic.

5. Disseminated sclerosis, multiple sclerosis.

6. Tumors (meningiomas, acoustic neuromas, pearly body tumors).

7. Exposure to cold.

DISCUSSION.

Undoubtedly the neurovascular mechanism, with the adjacent internal carotid artery and its branches, has a definite bearing on the cause of tic douloureux. The fact that the operation devised by Taarnhoj in 1952, showing that there is traction on the dura with constriction of the dural bands along the bony petrous ridge, is significant and may be connected with the neurovascular mechanism, apparently causing pressure on the Gasserian ganglion.

Emotional: After the inception of this excruciating pain the psychic element is unquestionably present. The grimaces, the fear, the anticipated torture are profound.

Foci of infection, especially dental and sinus in nature, and involvement of the temporomandibular joint from an arthritic standpoint, may be contributing factors. All sinuses and mouths should be X-rayed, regardless of whether or not they are edentulous. Probably over 20 per cent of all edentulous mouths have embedded tooth roots around which are infectious processes in most cases.

General systemic infectious disease may be an associated factor. It has long been known that multiple sclerosis is oftentimes found in cases of trigeminal neuralgia; and, of course, tumors (meningiomas, acoustic neuromas, pearly body tumors) and aneurysms of the carotid and other intracranial masses may be involved.

Although it is a controversial issue, changes in heat and cold, exposure to cold, both externally and intraorally, have a bearing on the inception of this disease.

DIFFERENTIAL DIAGNOSIS.

The following are the most frequently found conditions which simulate trigeminal neuralgia, and which must be ruled out:

1. Sinusitis.

2. Dental involvement (embedded third molars), edentulous mouths.

3. Glossopharyngeal neuralgia.

4. Temporal arteritis.

5. Nasal septal spur neuralgia.

6. Sphenopalatine ganglion neuralgia.*

7. Horton's histamine cephalalgia.

8. Costen's syndrome—temporomandibular joint.

9. Furunculosis of the ear canal.

10. Geniculate ganglion neuralgia.

11. Glaucoma.

12. Intracranial tumors.

13. Trigeminal neuralgia—not tic douloureux (Dandy).

14. Migrainous neuralgia—*Harris.*

Sinusitis: Greatest pain is early in the morning and on one side, it subsides toward noon and may be bilateral.

Glossopharyngeal Neuralgia. Love states: "If the patient complains of spasmodic excruciating pain that extends from the pharynx and tonsillar fossa on one side to the homolateral ear, and the pain is precipitated by swallowing, talking or yawning, the examiner can be rather certain that the patient has glossopharyngeal or IXth nerve neuralgia. If the examiner desires further proof of the diagnosis, the involved side of the pharynx and the tonsil may be cocainized and the patient given a glass of water. If on drinking, no pain is produced, the diagnosis is confirmed. Cocainization of the throat has no effect on neuralgia of the third branch of the Vth nerve. Likewise, injection of alcohol into the third branch of the Vth nerve has no effect on glossopharyngeal neuralgia."

Temporal arteritis has a flushing and perspiration, and usually responds to steriod treatment. It may be associated with the temporomandibular joint. Most of the others in this

*Sphenopalatine ganglion neuralgia is a misnomer. This should be deleted from the literature.

differential diagnosis can be determined by careful anamnesis, together with X-ray, laboratory and other findings.

DIAGNOSIS.

1. Elderly patient (female) (edentulous).
2. Agonizing unbearable pain, paroxysmal in nature.
3. Trigger zone—ala nasi, upper lip, buccal mucous membrane.
4. Dietary deficiency and dehydration—lack of nourishment.
5. Seasonal exacerbation (Spring and Fall).
6. Careful anamnesis.
7. Patient's description of pain.
8. The grimaces and fear mentioned above.

QUESTIONABLE THERAPY.

When considering medical and temporizing surgical therapy which gives temporary relief we may lull ourselves into complacency and distort the true picture.

MEDICAL THERAPY.

All of the following have been tried from a medical standpoint and many articles show that in the hands of a certain doctor they seem to have proven efficacious. There is always a question as to whether the patient had a true trigeminal neuralgia.

1. Dilantin sodium.
2. Priscoline.
3. Histamines (vasodilators).
4. Nicotinic acid.
5. Stilbamidine isethionate (taken off of market).
6. Vitamin B_{12}, vitamin B_6.
7. Steroid therapy.
8. Ataraxoid—Tranquilizing preparations.
9. Narcotics and allied drugs.
10. Protamide.

TREATMENT: INJECTION THERAPY.

1. Procaine hydrochloride.

2. Absolute alcohol 95 per cent.

3. Supraorbital, infraorbital, mental.

4. Deep alcohol injection or infiltration maxillary and mandibular branches at base of skull.

5. In Gasserian ganglion contraindicated.

There are some hazards in connection with the injection of absolute alcohol 95 per cent into a nerve body or foramen, because of the possibility of sloughing tissues, involvement of the adjacent nerves, and alcohol has also been accidentally injected intravenously. There are many advocates of alcohol injection, but there is danger in injecting the Gasserian ganglion.

ALCOHOL INJECTION ADVANTAGES ARE:

1. Poor risk patient—elderly, cardiovascular-renal complications.

2. In younger patient to ascertain status and possibly spontaneous remission.

3. To confirm diagnosis (possible atypical neuralgia).

4. To ascertain premonitory warning of organic lesion.

5. To promote temporary freedom of pain—patient's general hygiene improvement.

ALCOHOL INJECTION DISADVANTAGES ARE:

1. Sloughing tissues.

2. Alcoholic neuritis.

3. Burning paresthesias.

4. Continued repeated injections.

5. Never do deep injection of first division.

6. Inadvertent subarachnoid infiltration.

SURGICAL THERAPY.

1. Peripheral—middle cranial fossa—centrally; *a.* Superficial foramina in face, nerve crushed or avulsed; *b.* transtemporal craniectomy—Gasserian ganglion middle cranial fossa; *c.* posterior sensory root—tractotomy (mesencephalon-medulla oblongata).

The branches of the trigeminal nerve may be cut either centrally in the Gasserian ganglion or at various branches, infraorbital, mandibular foramen, mental foramen, et cetera.

The transtemporal craniectomy devised by Taarnhoj, and exemplified by Love and Woolsey, has its advocates and certainly is to be commended.

The tractotomy operation involving the mesencephalon-medulla oblongata severing the posterior sensory root may be performed by careful neurosurgeons. The transtemporal retrogasserian sensory root section by Frazier and Spiller has probably been performed more than most of the others. I am not familiar with Kirschner's electrocoagulation of the Gasserian ganglion, but it sounds like it might have merit.

Jaeger's hot water injection through the foramen ovale with complete destruction has no untoward results; no anesthesia of the eye; no involvement of other nerves, and it may be a great contribution. Jaeger reports 130 patients in whom there have been no deaths, no facial paralysis, and no other complications. He states that this should be performed by a neurosurgeon. It is my opinion that the training in otolaryngology should include not only the ability to inject through the foramen ovale with hot water, but to resect the sensory root (maxillary and mandibular) of the trifacial nerve.

COMPLICATIONS—NEUROLOGIC DEFICIT.

1. Anesthesia of face (substitute for agonizing paroxysms of pain).

2. Corneal desensitization.

3. Lagophthalmos.

4. Abducens nerve palsy.

5. Fourth nerve (trochlear) paresis.

6. Herpetiform vesicles—eyelids.

7. Keratitis neuroparalytics.

8. Nerve deafness.

9. Paralysis of motor trigeminal and facial (usually temporary with facial but may be permanent with nerve deafness).

10. Diminshed secretions of tears.

Of the above, undoubtedly the corneal involvement through its desensitization with a subsequent keratitis is tremendously significant. Many of the others may be equally as significant, but they are found less frequently. In the corneal desensitization the ophthalmic branch, naturally, has been sectioned.

GENERAL CONSIDERATIONS.

1. Inadequately fed, dehydrated, nervous.

2. Apprehensive, pain-wracked, debilitated.

3. Subclinical dietary deficiency, prolonged bleeding time.

4. Protein-poor dehydrated patients—wounds heal poorly.

5. Blood electrolyte patterns abnormal.

6. Mental confusion—retraction of temporal lobe.

7. Blood urea nitrogen—fluid balance.

8. Liquid intake and urinary output.

9. Restlessness, apprehension, depression.

10. Barbiturates, adverse reaction—disorientation.

11. Considerate thoughtful attention from surgeons.

One must carefully evaluate each one of these and be a psychiatrist par excellence, a keen internist and equally capable surgeon, besides being a philosopher.

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AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

The American Association for Cleft Palate Rehabilitation will hold its Eighteenth Annual Convention at the Brown Palace Hotel, Denver, Colo., Thursday, Friday and Saturday, May 12, 13 and 14, 1960.

This Association is composed of medical, dental and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates.

TOTAL PAROTIDECTOMY.*

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New York, N. Y.

A great deal of progress has been made in surgery of the parotid gland since the turn of the Century. In the early editions of "Treves," that small but mighty compendium of surgical anatomical knowledge, the complete removal of the parotid gland was described as a surgical impossibility. Paradoxically, the progress that has been made since that time has been based on an erroneous concept of the structure of the gland, namely, that it is a bilobed organ.

Gregoire, in 1912, was the first to describe it as a bilobed organ. McWhorter, in 1917, supported this idea and indicated that the isthmus was located between the two main divisions of the facial nerve, namely, the cervico-facial and the temporo-facial divisions. Bailey, in 1941, developed the idea further and claimed that there was a natural plane of cleavage between the two lobes that was easily identifiable and in which the facial nerve was found. He further stated that the only portion of the gland that had to be divided was the isthmus, which he located between the two divisions of the facial nerve. Martin, in 1952, in commenting on the plane of cleavage, doubted that there was such an anatomical plane but retained the nomenclature of a superficial and deep lobe in his descriptions.

McKenzie, in 1948, basing his conclusions on anatomical studies, showed that the gland was not bilobed with a single isthmus, but that it had multiple isthmi; in fact, he compared the gland to a creeping vine, weaving itself into the meshes of a trellis, the facial nerve.

The concept of the bilobed structure of the gland with a natural plane of cleavage has dominated our thinking regard-

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ing this organ, and undoubtedly has been a great source of reassurance for many surgeons assaying a parotid dissection. One would feel more confident in beginning his dissection if he knew that there was a natural plane of cleavage that he was dealing with, rather than the concept of McKenzie, of a vine weaving itself around a trellis.

Patey, in 1957, reviewed the surgical anatomy of the parotid gland and on the basis of his surgical and anatomical dissections proposed a new concept of division of the parotid. He proposed an anatomical surgical plane that was based upon the anatomy of the large veins which transverse the gland from above downward. Th eposterior facial veins, starting with the superficial temporal vessels at the upper border of the gland, pass through the substance of the gland on a plane just immediately deep to the facial nerve, and end at the lower pole of the parotid as the anterior facial vein and the posterior facial vein. The veins establish a definite anatomical plane throughout the entire gland. Patey has called this the facio-venous plane, and by keeping superficial to it throughout the dissection, the cleavage develops between the superficial and deep portion throughout the gland.

The practical value of this concept is that by so splitting the gland the venous circulation is not interrupted during the dissection of the superficial portion. This greatly reduces the bleeding which is for the great part venous oozing, which occurs when the lower pole of the parotid is dissected first and the posterior facial vein is cut and tied, and the glandular tissue becomes congested due to venous stasis.

The second practical application of the facio-venous plane is that it permits a more accurate description of the operative procedure. Thus, Patey divides the gland into a superficial portion which he names the superfacial, and a deep portion which he names the subfacial. Each of these two major portions are then divided into an upper and lower half called the suprafacial and the infrafacial. The surgeon, then, in describing a partial parotidectomy can accurately define the amount and location of the tissue which has been removed and the portion which remains.

The term total parotidectomy is used to describe a complete removal of the parotid tissue. This may be indicated in benign conditions such as the cystadenolymphomatosum and also sialectasia, or a total parotidectomy may be done when an exposure of a mixed tumor arising in the deep portion of the gland requires removal of both the superfacial and the subfacial portion. The term radical has been applied to total parotidectomy when the facial nerve has been sacrificed in its entirety, and conservative when it is possible to remove part of the gland and preserve all or part of the facial nerve.

PAN-PACIFIC SURGICAL ASSOCIATION.

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 28 through October 5, in 1960.

All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by leading surgeons promises to be of interest to all doctors. Nine surgical specialty sections are held simultaneously.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

SPONTANEOUS CEREBROSPINAL OTORRHEA.

Report of a Case With Successful Surgical Repair.*

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Pasadena, Calif.

Cases of spontaneous cerebrospinal otorrhea are so rare that they may be considered a medical curiosity. I am presenting a case which occurred in a woman of 72 years. While many cases of cerebrospinal otorrhea, which followed trauma or operations, have been reported, only one reference could be found of a case at all similar. This was a case reported by O. R. Kline[1] in 1933. His patient was a man of 54 who suddenly began to drain clear fluid from an apparently normal ear on leaning over to pick up an object. He developed meningitis and died three days after the onset of drainage. The autopsy showed what was probably a congenital fistula that had begun to drain without apparent reason. A slightly similar case was reported by Nenzelius,[2] of Sweden; his case was a 15-month-old girl with a number of other congenital malformations. She was cured after three operations and three recurring attacks of meningitis. His search of the literature found one other case reported by Eschat[3] in 1897. I was unable to get a copy of this report.

Case Report. A white woman of 72 was first seen by me on July 1, 1955. There had been a painless, watery drainage for two months without intermission. Seven months before, there had been a very slight painless drainage for two days. Trauma and recent infections were denied. About 35 years before, she had had a painful infection of the right ear that had recovered after an incision of the drum, followed by drainage for a few days. She thought her hearing was normal, but the audiogram showed about 20 db loss for conversational tones in both ears. Examination showed a small central perforation of the right drum in the posterior inferior quadrant, through which a clear watery fluid constantly drained. Tests showed 75 mg. per cent of sugar, which indicated cerebrospinal fluid. X-rays showed both mastoids well pneumatized with some pneumatization of the petrous tips. There was slight clouding of the right mastoid, but no other abnormality was seen. The amount of drainage would vary from day to day. On one day 5 cc. were collected in an hour and a half; this was about average.

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Fig. 1. Laminographic X-ray of right temporal bone showing defect lateral to arcuate eminence.

At first, the patient could not be persuaded to consider an operation, but finally after five months of continuous drainage she entered the hospital. She had known for a long time she had severe nephritis and a hypertension of 200/100. Neurological examination was negative. Laminographic X-rays showed a possible defect in the tegmen on the anterior edge of the right temporal bone just lateral to and anterior to the arcuate eminence (see Fig. 1).

Exploratory operation of the anterior face of the temporal bone was done Nov. 1, 1955, by our neurosurgeon, R. H. Pudenz, M.D. A circular piece of the temporal bone about 3 cm. in diameter was removed, and the dura stripped from the anterior slope of the petrous bone. In this bone was found five or six crater-like structures, the largest of which was 5 or 6 mm. in diameter (see Fig. 2). The dura was rigidly attached to them, and when it was stripped away there was an opening made, through which cerebrospinal fluid escaped. In the largest of these craters was seen some soft gray tissue which was taken for biopsy. This was later

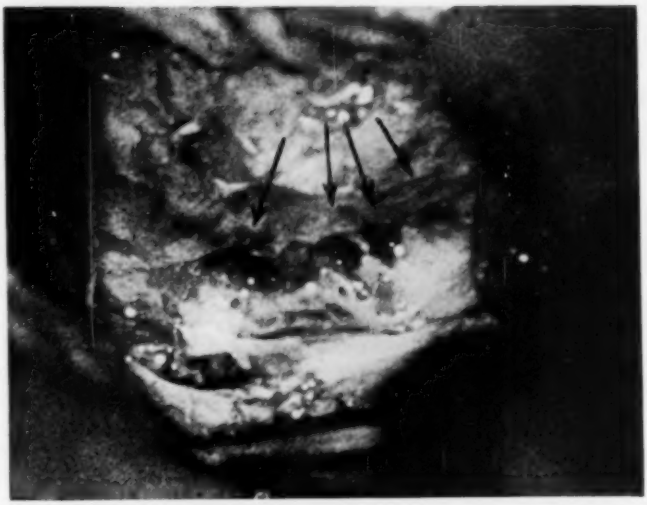

Fig. 2. Photograph taken at operation, showing "craters" in the anterior face of the temporal bone.

Fig. 3. Photograph taken at autopsy of "craters" in anterior face of the left temporal bone.

reported as nerve tissue of cerebral type. A flap of temporal muscle about 2 cm. in diameter was then cut and spread over the craters and sutured to the deep dura in three places. The incision was closed rapidly, as the blood pressure had dropped from 200/100 to 60/40. Transfusion was started at once, and the pressure came back to 160/90 before she left the room.

Postoperative Course. The patient recovered rapidly and there was never any more drainage from the ear. She died 18 months later following a thrombosis of the right middle cerebral artery.

Autopsy. Many of the previously described craters were found in the floor of both temporal fossae; more on the right side than the left (see Fig. 3). They were present from the anterior limits of the middle fossa to the extreme posterior portion of the temporal lobes. Minute projections of brain tissue, covered by dura, extended down into these craters.

Microscopic appearance: "Sections of the temporal and sphenoid bone show within what one would normally expect to be marrow spaces, nodular protrusions of glial tissue, somewhat resembling normal cortex but showing fewer cell bodies . . . Buds of tissue protruded off the larger masses to extend into lacunae. The dura appears to be perforated in these areas, and only a thin investment, apparently pia, covers the nervous tissue protrusions . . . There is no apparent reaction surrounding the masses."

Otherwise, the autopsy showed some softening of the brain, supplied by the thrombosed right middle cerebral artery, generalized arteriosclerosis, and chronic pyelonephritis. In his discussion the pathologist said "The extension of dura and brain substance into the temporal bone is unique, and we have not as yet run across a description of this condition anywhere."

DISCUSSION.

The temporal bones were removed and sent to Dr. Victor Goodhill's research laboratory. In sections of these bones A. A. Krajian, Ph.D., saw numerous clusters of cells which he thinks are kala-azar organisms. The patient's last physician remembered that she had been twice around the world many years ago, and at one time had a febrile infection diagnosed as malaria. Several pathologists have checked the same slides, and do not think that these cells are protozoons. I am inclined to agree with Drs. Shillam, Foord and Fisher, that the defects found in the temporal bones were developmental anomalies. Why one of them developed a leak down into the ear I do not know. I compliment our neurosurgeon, R. H. Pudenz, on finding the defect and successfully repairing it.

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL.

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-second Annual Spring Congress in Ophthalmology, Otolaryngology and allied specialties. The attendance was one of the largest in the history of the school with an attendance of 350 physicians and their wives. There were forty-two states, England, Canada and several foreign countries represented. There were twenty-one guest speakers, sixty lectures and closed circuit televised surgery during the five and one-half days of the Spring Congress. In 1960, the Thirty-third Annual Spring Congress will be held from April 4 through April 9.

VOICE TRAINING IN THE MANAGEMENT OF DYSPHONIA.*†

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The speech therapist will approach any problem of dysphonia in a manner somewhat different from the laryngologist. In the sense of effecting improved organic status, treatment is not the basic function of the speech therapist; it is his task to work within whatever limitations are imposed by organic problems and to help the patient to achieve the most acceptable function with the least strain on his physical equipment. Organic equipment may become healthier as the result of relief from the strain of misuse, but improvement of function remains the principal goal. Our remarks will be concerned with three things: characteristics of vocal function found frequently among patients with voice problems, general approaches to these problems within the discipline of speech therapy; and considerations in the prognosis for improvement through voice training.

Faulty vocal function cannot be separated from the human being who directs the function. The human voice itself is one of the best indices of the way the human feels. It expresses the feelings of the moment as well as the perennial patterns of adjustment of the individual. There is no reason here to dichotomize any complex aspect of human behavior into that labelled "functional" or "organic." The functional versus organic breakdown must properly be considered as a continuum, with the clear organic deficiencies such as dysphonias resulting from the surgical removal of all or a portion of a vocal fold, or vocal fold paralysis at one end, and the

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clearly functional problems such as hysterical dysphonia and persistent prepubital voice at the other end.

The majority of voice problems will be found to fall within these extremes. Cause and effect are so intricately interwoven that it is usually impossible to solve the "which comes first" riddle. Vocal folds which are irritated by acute or chronic infections or allergic reactions will take abuse more readily; on the other hand, vocal folds which are irritated constantly by misuse are more apt to suffer organic changes.

It is not our purpose to catalog the myriad characteristics of faulty vocal production to be found in the total population of patients with voice problems. The most common are: *1.* harsh initiation of vocal tones; *2.* faulty pitch placement; *3.* restriction of pitch range, and *4.* imbalance of vocal resonance. Almost inevitably, these characteristics will not occur singly, but together, in varying degrees.

Harsh vocal attack is probably the single factor most commonly found. It is usually heard as a "glottic shock" or "glottic stroke," in which vowel sounds are initiated with a click and give a harsh, rattling quality to the voice. A number of primary factors are present in the production of the harsh vocal attack, but these all lead to the secondary factor of tension within the laryngeal musculature. As a consequence of this tension, the vocal folds adduct to initiate phonation with a sharp catch rather than producing a full, smooth tone.

In the second characteristic, faulty pitch placement, the architecture of an individual's vocal mechanism—its component parts, their size, shape, tissue characteristics, etc., will generally determine the most efficient or optimum pitch range at which the individual's voice will function. Problems may occur when the individual consciously or unconsciously chooses to use the voice habitually outside or at one of the extremes of his natural pitch range.

The restriction of pitch range is closely related to faulty pitch placement and is also associated with tension. In this problem, the normal pitch variations which produce the inflections of ordinary discourse do not occur or are limited in extent, and the voice becomes flat and monotonous. Usually,

only the three or four tones at the lower end of the pitch range are used. The use of this characteristic is common on the stage, when an actor attempts to convey the concealment of emotional response.

Resonance of the human voice occurs chiefly in the pharyngeal, oral, and nasal cavities. In the normal voice, some degree of participation of each of these resonators is present throughout phonation; however, it must be in "balance." Problems occur when resonation in one of these cavities is excessive or when it is diminished. The accentuation of pharyngeal or nasal resonance is common among dysphonic patients.

The following sequence of objectives in voice training or re-training are summarized briefly, even though many variations result from the idiosyncrasies of each individual patient and his problem:

1. Simple initiation and production of a full tone within the optimum pitch range, clear in quality, and in which the various resonance components are in proper balance.

2. The ability to utilize this vocal tone spontaneously, with auditory and kinesthetic monitoring, and with recognition of the differences between the new tone and the habitual manner of vocal production.

3. The ability to utilize this tone in increasingly longer language units—from the syllable, to the word, to the phrase, to the sentence, and to longer passages.

4. The use of more acceptable voice in the activities of daily living.

Many methods are used in the achievement of improved vocal tones. These techniques are directed at providing adequate breath support for vocalization, determination of the point at which abrupt adduction of the vocal folds occurs, replacement of this sharp adduction with smoother vocal fold contact, and restoration of balance in resonation.

Retraining of vocal pitch has been deliberately omitted from discussion here, since faulty pitch placement is usually

treated as a secondary rather than a primary factor. Although it is mentioned as a common trait among patients with voice disorders, faulty pitch placement is usually only one evidence of faulty phonation. Mere pitch change does very little to change the basic fault in vocal production. When a clear, fluid vocal tone is achieved, the pitch usually automatically changes to the approximate optimum. If the problem of pitch is attacked separately, it is more easily approached after improved phonation is achieved.

In the therapy sequence, emphasis is placed on assisting the individual to recognize exactly what he is doing in the production of his voice. When confronted for the first time with a tape recording of his own voice, almost any person will deny its veracity; this denial is even stronger in the presence of a voice problem.

We monitor our voices auditorially; the deterioration of voice quality commonly heard in a deafened adult will offer ample evidence of this. We also monitor our voices kinesthetically. This can be demonstrated by introducing sufficient masking noise into the ears to block out all side tones. Usually, even though volume increases, the voice quality changes are minimal. The relationship between muscular tension and voice disorders is a pertinent one here. Tension can be perceived only as a kinesthetic experience. From the beginning of the training period, the therapist must stress constant and accurate monitoring of each vocal production.

The process of assisting an individual to use this improved vocal production in his daily discourse is more complex than an admonition. Of primary importance is the individual's willingness to change and his ability to focus his efforts on the amelioration of the voice problem. The surgeon requires only the physical presence of a patient for the treatment of a problem; but the speech therapist can offer the patient only a variety of activities which may effect functional improvement and act as a guide. Changes are effected by the patient himself. Once improved phonation is achieved in the therapy session, it is the sole responsibility of the patient as to whether it is carried outside of the speech clinic. The speech therapist can assist the patient in recognizing those factors which pre-

vent his use of improved phonation in the world outside of the clinic, in the hope that the patient can work out his own solutions to the interferences. When these interferences represent complex neurotic manifestations, the patient should be referred to a psychotherapist.

Laryngologists have often asked for suggested techniques which might be used during an office visit to effect improved vocal usage. This is difficult. The referral from laryngologist to speech therapist, and back again to the laryngologist, can be a very clumsy process. There would be many advantages to combined medical treatment and voice training by one individual; however, the problems involved are very practical. The alteration of firmly fixed vocal habits often requires semi-weekly contact between therapist and patient for a period ranging from three to six months, or more. The schedules of most laryngologists seldom permit this amount of time to be given to any single patient.

When dealing with the results of any procedure as difficult to objectify as voice training, we can only estimate prognosis on the basis of our own experience. Our views of prognosis are presented here on the basis of a review of a series of 50 patients with whom we have worked during the past five years. The patients reviewed were selected only on the basis of the criteria that continued contacts with the speech clinic were maintained for a period of at least six months and that a reexamination by the referring laryngologist was available at least six months following termination of voice training. In several instances reexamination one year following termination of therapy were available. Voice training was considered successful when a more acceptable voice was achieved and maintained, and when there was no evidence of misuse on laryngeal examination at the six months' recheck.

Among this relatively unselected clinical population, 25 patients achieved success according to the stated criteria. Pubblished reports by Williamson[1] claim success with 61 out of 69 patients; Peacher[2] reports success with five out of six patients with contact ulcers; and Gardner[3] describes success in the treatment of all 37 of his patients with what he calls

"executive's dysphonia." It is our impression that in routine clinic practice the degree of success met is seldom as high as claimed by these writers.

Further examination of our patient records leads to these additional conclusions regarding prognosis:

1. Barring irreversible pathology or structural anomaly of the vocal mechanism, the reported condition of the larynx is an unsatisfactory measure of prediction of ultimate success in retraining. The prognosis remains about the same whether the larynx appears essentially normal, whether inflammation is present, or whether nodes, nodules, contact ulcers, etc., have developed.

2. The adult patient's age does not predict his probable success in retraining, except for the voice changes of senescence. The patient of 50 years may be as amenable to voice change as the patient of 20 years; however, pre-adolescents who have developed dysphonias as the result of vocal abuse present extremely difficult problems for voice training. The degree of concentration, critical listening, and self-supervision which the process demands can seldom be expected in childhood.

3. Voice disorders which are ordinarily associated with misuse are not found exclusively among people in professions which demand constant voice use (*e.g.*, law, the ministry, teaching, etc.). On the other hand, the fact that voice usage is essential to an individual's vocation is no insurance that he will succeed in voice retraining—in the author's experience, again only approximately one-half achieve lasting improvement.

4. Although a somewhat higher number of women than men come to our clinic seeking voice therapy (the ratio is four to three in our population) significantly fewer women than men achieve success in voice training. It is generally believed that most hysterical conversions have a somewhat higher incidence among women. It is possible, therefore, that more of the voice disorders found among women are manifestations of complex problems of adjustment, and as such, are given up much less readily.

5. No other specific prognostic factors, such as thyroid disorders, foreign dialect influence, or the length of time since the initial awareness of the voice problem, seem to yield information of any certainty in the prediction of potential success in voice training.

It is probable that the really significant characteristics of a patient which will predict whether voice training will be successful are those which cannot be objectified. An ideal candidate is an individual who is sufficiently intelligent and secure to be insightful, who can learn to listen critically, who is able to approach his problem directly, and who is willing to direct real effort toward the difficult task of changing long-standing habit patterns. Conversely, the least likely candidate is the patient who finds insight difficult or threatening, who cannot permit himself to listen honestly, who may protect himself by scattering his attention, and who cannot muster his efforts to work diligently for improvent. We are again confronted with two ends of a continuum; between these ends falls the majority of the patients.

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THE RADIOLOGIC STUDY OF SOME NORMAL AND ABNORMAL SWALLOWING MECHANISMS; ASPIRATION PHENOMENA AND CRICOPHARYNGEUS SPASM.*†

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New York, N. Y.

Advanced carcinoma of the larynx, hypopharynx and thyroid are frequently associated with dysphagia and aspiration phenomena. The roentgenologist is requested to determine whether the aspiration is due to fistula formation or occurs through the normal laryngeal channel. In order to make this differentiation, an adequate roentgenographic visualization of the laryngopharynx during the second stage of swallowing is required. Routine conventional procedures are usually found inadequate because of the rapid sequence of events in this region during deglutition. Single, sporadic exposures of the barium-opacified laryngopharynx during swallowing do not yield the desired information concerning the abnormal mechanism involved. For this reason it was decided to use a somewhat more elaborate technique in the study of deglutition. A Fairchild rapid changer was employed which yielded 9x9 inch roentgenograms of the laryngopharynx up to a rate of two per second.

The results with this apparatus were found highly satisfactory after some experience had been obtained. The 9x9 inch films produced life size images permitting analysis in very fine detail of the various portions of the larynx and hypopharynx during swallowing. Such detailed visualization was found essential in obtaining a proper understanding of the mechanisms involved in normal and abnormal swallowing. The other instrument which has been employed recently in

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the study of deglutition is the cineradiograph, usually carrying 16 mm. film. The latter apparatus produces pictures at a faster rate, but the detail is very much less than that obtained with the 9x9 inch changer. It would appear that the advantages of greater detail may more than offset the greater speed factor of the cineradiograph in the proper interpretation of events in the laryngopharynx during deglutition.

ASPIRATION PHENOMENA.

In order to understand the radiographic features of abnormal swallowing and aspiration it is necessary to acquaint oneself with the radiographic appearance of the normal anatomy and physiology of swallowing. The entrance from the laryngopharynx into the laryngeal vestibule (*i.e.*, the laryngeal aperture) is bounded in front by the epiglottic rim, on each side by the aryepiglottic folds, and posteriorly by the arytenoid cartilages sliding on the cricoid lamina (see Fig. 1). Lateral to the aryepiglottic folds and side walls of the vestibule are the pyriform fossae. Our studies indicate that these structures, which rim the laryngeal aperture, form the primary and essentially the sole barrier preventing the passage of fluid from the hypopharynx into the larynx during the second stage of swallowing. In our experience to the present time, the true and false cords have never performed as competent safeguards against aspiration. In each instance where the barium suspension was able to penetrate the protection ring about the laryngeal aperture and reach the cords, the fluid passed the rima glottidis and entered the trachea. The process of swallowing is best studied radiographically with the rapid changer in the lateral view. In this position there is the least overlap and barium obscuration of the epiglottis, aryepiglottic folds, arytenoid cartilages, thyroid and cricoid cartilages, bands and cords.

An analysis of the radiographs made with the rapid changer at two per second during barium-suspension swallowing, reveals the following sequence of events in the normal laryngopharynx. The erect epiglottis, situated behind the posterior aspect of the tongue, acts as a partial barrier and dam in the midline against the passage of fluid from the mouth into the

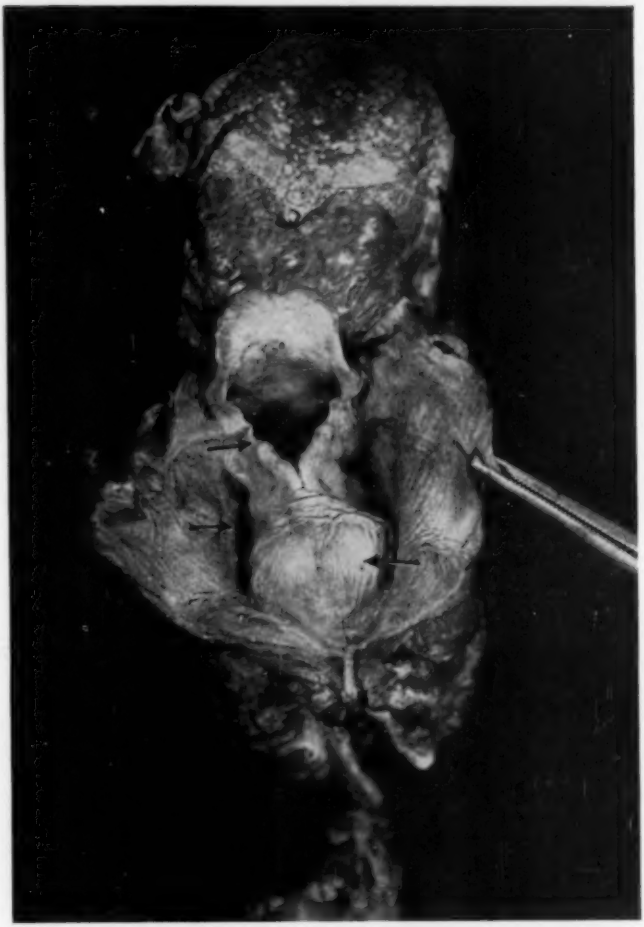

Fig. 1. Larynx and laryngopharynx seen from behind. Laryngeal aperture well shown. Right upper arrow at aryepiglottic fold; right lower arrow at pyriform recess; left arrow at cricoid lamina.

Fig. 2. Swallowing of small amounts of fluid. Note contrast material spilling around sides of erect epiglottis (upper arrow) running down the pyriform recess trenches on each side (middle arrow) into the esophagus (lower arrow). The aryepiglottic folds remained straight and the laryngeal aperture is open. There is no spill into the vestibule.

upper pharynx. Fluid may enter the pharynx in either of two ways. If a small quantity of fluid is ingested it is insufficient in amount to pass over the centrally placed erect epiglottis. The fluid flows on each side of the epiglottis and enters the pharynx laterally, passing over the pharyngo-epiglottic folds and flowing in the pyriform recesses on each side of the laryngeal vestibule. Because the pharyngeal side walls approximate the laryngeal vestibule at this time, the pyriform recesses assume the form of shallow troughs which channel the small amounts of fluid from the lateral margins of the epiglottis down and around the laryngeal vestibule into the postero-inferior hypopharynx and directly into the esopha-

gus (see Fig. 3-a). The laryngeal aperture and airway do not close. The two lateral shallow pyriform recess trenches are adequate to direct all of the small amount of ingested fluid into the esophagus, and none of the fluid spills over the troughs into the open laryngeal aperture airway.

On the lateral view radiographs, following the swallowing

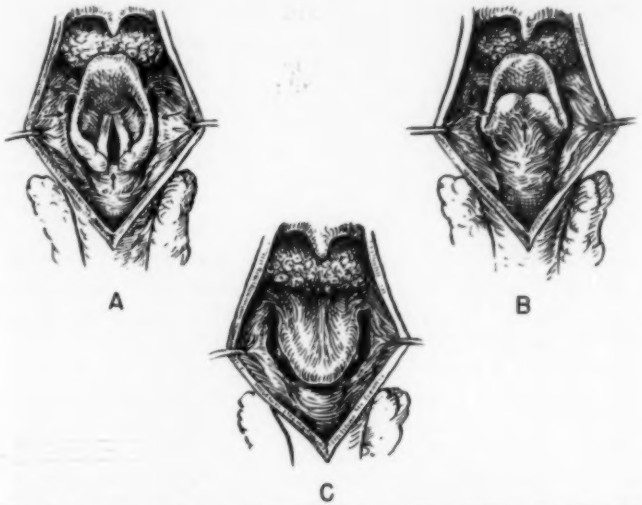

Fig. 3. A.—Open laryngeal vestibule in resting stage. Upper arrows point to pyriform recess trenches on each side of the aryepiglottic folds. Lower arrow at open interarytenoid commissure. B.—Closing of laryngeal vestibule just prior to passage of bolus. Lateral arrow points at aryepiglottic fold furrow caused by bending of this fold with elevation of arytenoids. Medial arrow points at closed interarytenoid commissure between the elevated arytenoid cartilages which have been pushed against the back of the epiglottic plate. C.—Epiglottis has been turned backward and downward over the elevated arytenoids by passage of the large bolus over this structure during swallowing.

of small quantities of barium suspension, the pyriform recess troughs may be visualized as thin opaque bands extending downward, around and backward from the epiglottis to encircle the open laryngeal aperture and join at the esophagus below the larynx (see Fig. 2). The suspension does not spill over the aryepiglottic folds into the vestibule.

When large amounts of fluid are ingested in a single swal-

low, the bolus entering the pharynx is too large to be completely channelled in the pyriform recess troughs, and the fluid spills over the aryepiglottic folds onto the larynx. The second, and more important, protective mechanism is then utilized.

The laryngopharynx, in protecting against aspiration during the second stage of swallowing of such a large fluid bolus, follows a comparatively rigid pattern of actions in fixed sequential relationships (see Fig. 3). As the fluid bolus reaches the back of the mouth there is a strong action of the pharyngeal and extrinsic laryngeal muscles which results in a moderate elevation of the hyoid bone and marked elevation of the cricoid and thyroid cartilages so that the latter closely approximate the hyoid bone. As the cricoid cartilage is elevated, the arytenoid (and adjacent corniculate and cuneiform) cartilages which rest on the cricoid lamina are also raised. While ascending, the two arytenoid cartilages approximate each other, closing off the arytenoid commissure. At the peak of their ascent the now-adjacent arytenoids are jammed against the posterior surface of the epiglottic plate below its superior rim, thus effectively sealing off the laryngeal vestibule from the laryngopharynx. With the elevation of the arytenoids toward the epiglottis, the aryepiglottic fold on each side bends at its center so that the superior half of the fold meets the inferior half and seals off the lateral margin of the laryngeal aperture from the lateral laryngopharyngeal passageway.

At the line of apposition of the upper and lower halves of the aryepiglottic fold on each side a thin furrow is formed (see Fig. 3-b). Frequently, a small amount of barium suspension will become lodged in each of the two furrows and appear as two thin, opaque lines projecting forward from the elevated arytenoid cartilaginous mass areas in the lateral roentgenograms made during swallowing (see Fig. 4). The manner of closure of the laryngeal aperture described above was seen in all the normal cases studied. A purse-string-like closure, thought by some to be the mechanism employed, was not observed. Anatomically, the comparatively broad rigid expanse of the epiglottic cartilaginous plate, the thick masses of the arytenoids and the exceedingly poor musculature at the rim

Fig. 4. Lateral view of laryngopharynx following barium opacification. Upper arrow at turned-down epiglottis; middle arrow points to opaque lines of barium in the aryepiglottic fold furrows; lower arrow at filling defect caused by elevated arytenoid cartilages.

of the laryngeal aperture make almost impossible a purse-string closure wherein all elements contract in size significantly.

Following the preparatory elevation of the larynx and closure of the laryngeal aperture, the large fluid bolus in the back of the mouth is now pushed down into the laryngopharynx. Part of the fluid passes on each side of the epiglottis into the lateral pharyngeal passageways and then downward, posteriorly and medially, into the esophagus. The fluid is prevented from entering the lateral aspects of the laryngeal aperture by the closed aryepiglottic fold furrows. The major portion of the fluid passes directly over the erect epiglottic tip. The strongly propelled large fluid bolus causes the epiglottic rim to be bent backward on itself over the closed laryngeal aperture, and it comes to lie over the elevated mass of the arytenoid cartilages which have been pushed against the back of the epiglottic plate (see Fig. 3-c). This backward bending of the epiglottis is not an essential factor in the prevention of aspiration into the laryngeal vestibule, since we have observed a number of cases in which the epiglottis has been excised with no resulting postoperative aspiration phenomena; furthermore, in a number of normal cases observed by us, the epiglottic tip has failed to bend backward and downward and has remained erect throughout the second stage of swallowing. In these cases, also, there has been no aspiration.

After the bolus of fluid passes the laryngopharynx and enters the esophagus the epiglottic tip assumes its erect position; the larynx and cricoid cartilages descend to their resting positions; the aryepiglottic folds straighten out, and the laryngeal aperture is restored to its usual open status so that normal respiration may be resumed. Small amounts of residual fluid in the laryngopharyngeal region are easily handled by the two lateral pyriform recess channels between the open aryepiglottic folds and their respective pharyngeal side walls. When the next fluid bolus reaches the back of the mouth the entire process is repeated.

For a better understanding of the swallowing mechanism it has been found useful to summarize the second stage normal swallowing sequence into the following sub-stages:

1. Closure of the center of the laryngeal aperture—brought about by elevation of the cricoid and arytenoid cartilages and pressing of the arytenoid cartilages against the back of the epiglottis.

2. Closure of the sides of the laryngeal aperture—brought about by bending and closing of the aryepiglottic folds with the formation of the aryepiglottic fold furrows anterolaterally.

3. Passage of the bolus with downward and backward bending of the epiglottic tip.

4. Reopening of the laryngeal aperture—effected by the return of the epiglottic tip to its normal erect position and the descent of the cricoid and arytenoid cartilages.

We have observed that abnormal aspiration into the tracheobronchial tree occurs by a number of different mechanisms. These may be classified as follows:

I. Aspiration Through the Laryngeal Aperture.

A. No intrinsic organic abnormality in the laryngopharynx. Abnormalities probably of neurogenic origin.

1. Faulty timing of the stages with respect to passage of the bolus.

2. Inability of the various furrows to close completely and form an effective seal against the leakage of fluid past them.

3. Inability of the various furrows to remain closed for the entire period during which the swallowed fluid is passing by.

B. Organic disease present in laryngopharynx; neoplasms, infiltrations, etc.

1. Inability of the folds to close properly and completely because of mechanical obstruction of a mass.

2. Inability of folds to close properly because of limited mobility of arytenoids or epiglottic folds due to neoplastic or inflammatory infiltration.

3. Inability of folds to close properly because of surgical procedures on the laryngopharynx.

II. Aspiration Through Abnormal Openings. Fistulous Tract Formation. These May Occur Anywhere in the Neck or Thorax.

These may occur anywhere in the neck or thorax.

In the absence of organic disease locally, aspiration is usually the result of functional disturbances and may be of neurogenic origin. Faulty timing with respect to the passage of the fluid bolus is commonly encountered. As an example, the cricoid and arytenoid may be tardy in rising so that the fluid enters the laryngeal aperture before it can close completely. Another similar mechanism is the premature descent of the arytenoids so that the aperture opens before all of the fluid bolus has passed it, with the result that some of the fluid enters the vestibule and is aspirated. In other instances the timing may be satisfactory, but the apparently normal aryepiglottic folds or arytenoids may fail to remain closed and act as an effective seal against the entrance of fluid into the larynx.

The second category of cases includes those with bulky neoplasms or infiltrating lesions which block adequate closure of the laryngeal aperture mechanically or inhibit the normal closing actions of the aspiration barrier. The presence of these abnormalities is usually apparent clinically and on the preliminary radiographic studies of the neck. In this group also belong the cases with aspiration following extirpative surgery other than laryngectomy for various tumors of the supraglottic larynx. It has been observed that removal of the epiglottis is often not followed by abnormal aspiration phenomena. This is not unexpected, since the closed arytenoid cartilages can still be brought up and pressed against the back of the tongue or residual epiglottic plate to seal off the laryngeal aperture effectively. Further investigation of postoperative cases is planned; nevertheless, it is presently believed that preservation of the aryepiglottic folds during surgery is important in preventing aspiration. Where these folds are absent or incapable of complete closure, aspiration

occurs. As yet, no instances have been observed where the true and false cords have been able to act as a satisfactory substitute for the laryngeal aperture components in the prevention of aspiration. In all instances thus far, whenever contrast material reached the rima glottidis it went through and entered the trachea; however, further investigation of

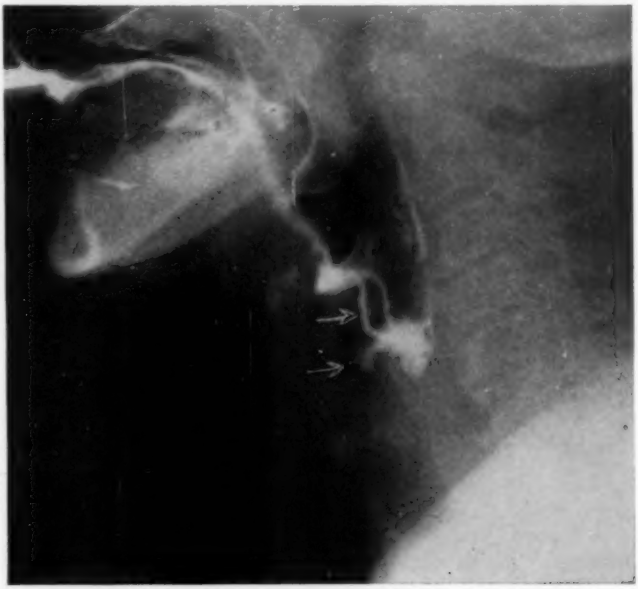

Fig. 5-a. Case 1. Premature descent of aryepiglottic folds with opening of laryngeal aperture. Contrast material spills over the arytenoid cartilages into laryngeal vestibule (lower arrow). Upper arrow at contrast material in pyriform recess trenches.

the competency of the glottic elements in preventing aspiration, particularly in the postoperative cases, is being continued.

The final group of cases is those in which fistulous tracts exist between the hypopharynx or esophagus and the upper tracheobronchial tree. These abnormal connections may occur in the neck or thorax. In proving their existence it is necessary to demonstrate the fistula itself and to show that

there is no aspiration through the normal laryngeal aperture into the vestibule.

Several brief case reports will illustrate a number of the varieties of aspiration phenomena described above.

Case 1. Male, aged 72, suffered a cerebrovascular accident, following which he began to aspirate ingested fluids. Direct laryngoscopy at the

Fig. 5-b. Contrast material which has entered the laryngeal vestibule (upper arrow) is seen to descend through the rima glottidis (lower arrow) into the trachea.

Francis Delafield Hospital revealed no organic abnormality in the hypopharynx or larynx. Barium swallow studies with the Fairchild rapid changer demonstrated that the cricoid and arytenoid cartilages rose normally and that there was adequate closure of the laryngeal aperture at the time that the main fluid bolus was passing by; however, the arytenoids and cricoid cartilage descended prematurely before all of the bolus had passed. As a result, the laryngeal aperture opened prematurely, and contrast material spilled over the partly descended arytenoids into the laryngeal vestibule and down into the trachea (see Fig. 5). This case represents a well defined example of aspiration associated with faulty timing without local organic disease.

Case 2. Female, aged 54, was admitted to the Francis Delafield Hos-

pital with a documented carcinoma of the thyroid and extensive metastases in the neck. Aspiration phenomena and dysphagia had recently appeared. A lateral roentogenogram of the neck revealed marked soft tissue thickening due to the extensive cervical metastases. Barium swallow studies with the rapid film changer showed the timing mechanism to be normal throughout the swallowing cycle; however, the aryepiglottic fold furrow did not remain tightly sealed, and contrast material was seen to leak through the incompetent furrow anteriorly as the barium bolus filled the laryngopharynx during the second stage of swallowing. The barium leaking through the incompetent furrow entered the vestibule and descended through the glottis into the trachea (see Fig. 6). This case, therefore, represents an example of inadequate closure of the

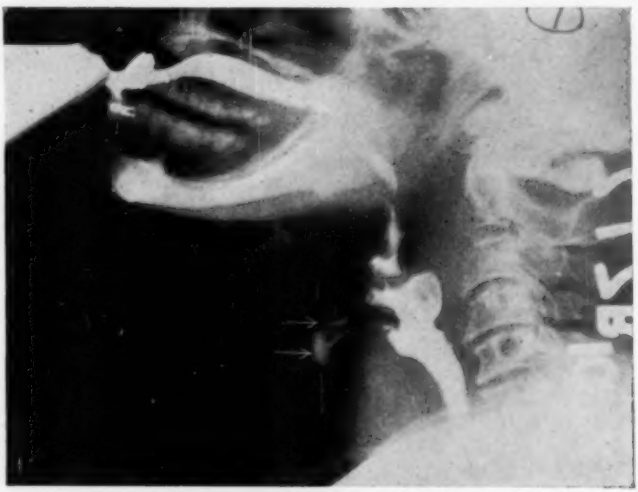

Fig. 6. Case 2. Aryepiglottic fold furrow is incompetent anteriorly and contrast material enters laryngeal vestibule just above ventricle (upper arrow). Some barium suspension already in trachea (lower arrow).

laryngeal aperture in one site due to mechanical and/or neurogenic factors associated with extensive neoplastic disease.

Case 3. Male, aged 62, was admitted with a documented carcinoma of the lung and markedly enlarged lymph nodes in both sides of the neck due to metastases. Shortly before admission, increasing dysphagia appeared, but there had been no aspiration phenomena. Roentgenographic examination of the neck showed the presence of large soft tissue masses due to the metastatic nodes. Barium swallow studies revealed marked deformity of the laryngopharynx lumen and deformity of the aryepiglottic fold furrows because of the enlarged cervical nodes; nevertheless, the timing of the actions of the laryngopharynx was normal, and the seals remained competent. As a result, aspiration did not occur (see Fig. 7). The dysphagia was due to the huge nodes which distorted and partially obstructed the laryngopharynx.

Case 4. Male, aged 55, was admitted with a huge carcinoma of the larynx which had caused complete obstruction of the airway and previously required tracheostomy. Aspiration of ingested fluid had appeared during the past six weeks. On roentgenographic examination in the lateral view the huge tumor occluding the larynx and the tracheostomy tube were evident. Barium swallow studies showed none of the opaque mixture to enter the occluded larynx; however, a well defined fistulous tract was demonstrated between the upper cervical esophagus and the trachea just above the entrance of the tracheostomy tube (see Fig. 8).

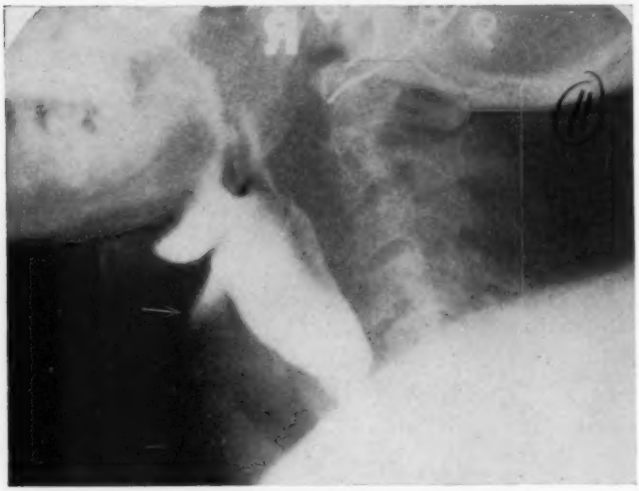

Fig. 7. Case 3. Aryepiglottic fold furrow deformed by large neck masses, but competent (arrow). No aspiration.

DYSPHAGIA DUE TO CRICOPHARYNGEUS SPASM.

One of the less common causes of dysphagia is spasm of the cricopharyngeus muscle. This muscle represents the lowermost transverse fibers of the inferior constrictor of the pharynx and lies immediately above the pharyngo-esophageal junction. The cricopharyngeus acts as a sphincter and normally is in a state of contraction during respiration. This closes off the esophagus from the hypopharynx, prevents the swallowing of air and directs the inspired air into the larynx and trachea. Our observations indicate that increasing the depth of respirations results in an increased degree of con-

traction of the cricopharyngeus, presumably to insure prevention of air from entering the esophagus. The most marked contraction of the muscle may be seen during a modified Valsalva maneuver, in which expiration is markedly obstructed by complete closure of the nares and almost complete closing of the lips. As a result, the cheeks bulge, the hypopharynx becomes distended with air, and the cricopharyngeus goes into marked tonic contraction. On the other hand, dur-

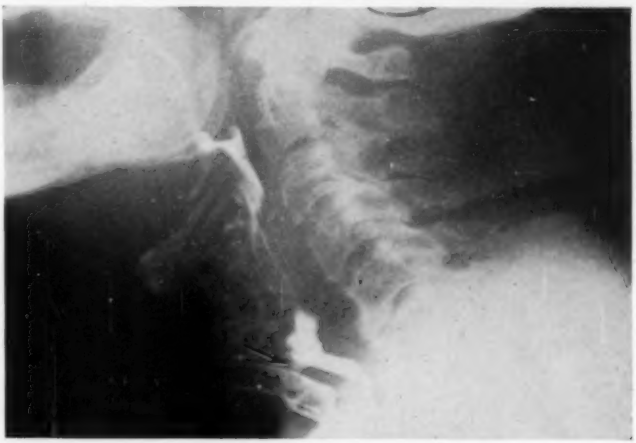

Fig. 8. Case 4. Larynx completely occluded by huge neoplasm. Fistulous tract present between cervical esophagus and trachea near entrance of tracheostomy tube (arrow).

ing swallowing the cricopharyngeus relaxes completely so that ingested material may pass from the hypopharynx into the esophagus without difficulty.

Radiographically, the cricopharyngeus is best demonstrated on the lateral view of the neck after a barium swallow and during the modified Valsalva maneuver described above. Sufficient barium residue remains in the hypopharyngeal region to delineate the thickened, contracted muscle bundle. The muscle lies immediately below and posterior to the lamina of the cricoid cartilage. Its main muscle mass is situated pos-

terior to the lumen of the inferior end of the hypopharynx and immediately in front of vertebral body C-5 or C-6. When it contracts, a sharply localized, smooth, rounded indentation is seen in the posterior aspect of the barium outlined lower hypopharynx just above the esophagus (see Fig. 9-a). No indentation of the anterior wall of the lower hypopharynx due to cricopharyngeus contraction is observed, since there is

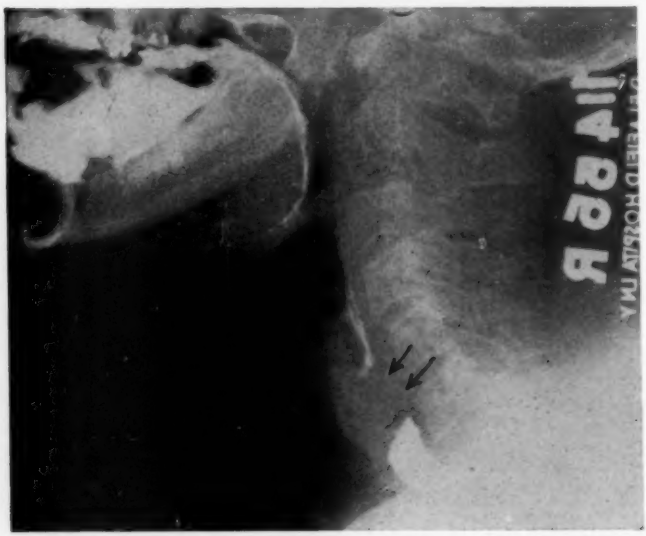

Fig. 9-a. Contraction of cricopharyngeus muscle during modified Valsalva maneuver. Note local forward displacement of barium column by contracted muscle band (arrows).

no significant number of muscle fibers between the lower hypopharynx and the upper trachea in the subglottic region. During swallowing the muscle usually relaxes completely, and the barium suspension passes from the hypopharynx into the esophagus with no or only minimal indentation on the barium column in the lower hypopharynx (see Fig. 9-b).

At times, the cricopharyngeus mechanism is deranged. Instead of normal relaxation during swallowing, the muscle

remains contracted. This interferes with the passage of ingested material from the hypopharynx into the esophagus and causes dysphagia. The etiology of the syndrome is obscure. No organic abnormalities have been found locally in the hypopharynx, and no associated organic abnormalities have been found elsewhere.

We have observed the syndrome in two cases in which re-

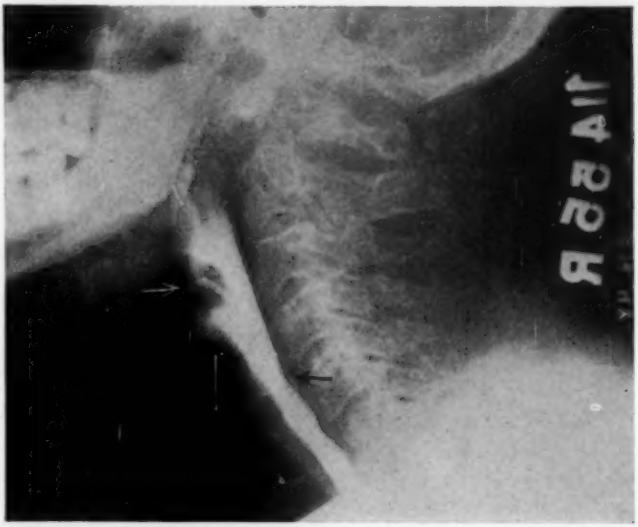

Fig. 9-b. Normal swallowing. Note complete relaxation of cricopharyngeus muscle (lower arrow) and uniform diameter of barium column. Incidentally, aryepiglottic fold furrow well shown (upper arrow).

current attacks of mild to moderate dysphagia were experienced for several years. The condition was not progressive, the attacks did not increase in severity and the patients' general health was not affected. There was no weight loss. One of the patients was a male; the other a female. The former thought that the dysphagia might be related to periods of mental upset. The second patient could not definitely relate the recurrence to emotional disturbances.

The radiographic appearances of both cases were very similar. In the lateral views, made with the rapid changer during barium swallows, each case showed a sharply localized, marked, smooth indentation on the posterior aspect of the barium-filled hypopharynx in the region of the cricopharyngeus muscle. The laryngo-pharynx cephalad to the indenta-

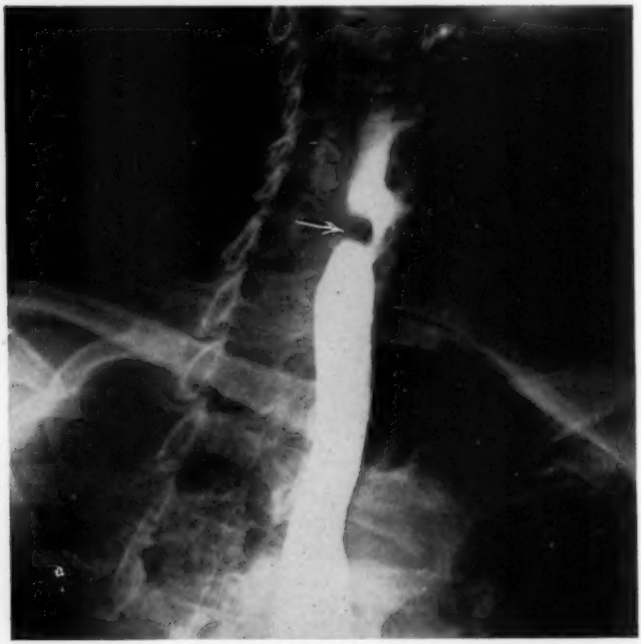

Fig. 10-a. Cricopharyngeus spasm. Note smooth indentation (arrow) on posterior aspect of barium column by contrasted muscle.

tion was slightly distended, but otherwise normal. The margins of the barium-filled pharynx were smooth and straight throughout, except at the cricopharyngeus site. No other soft tissue swelling was observed elsewhere, and there was no mucosal irregularity or ulceration. The anterior margins of the hypopharynx showed no abnormal indentations (see

Fig. 10). In the frontal views, a short zone of smooth narrowing, about 1 cm. in length, was noted between the slightly distended lower border of the hypopharynx and the upper end of the esophagus (see Fig. 11). The narrowing appeared due to the constriction of the barium column by the spastic cricopharyngeus.

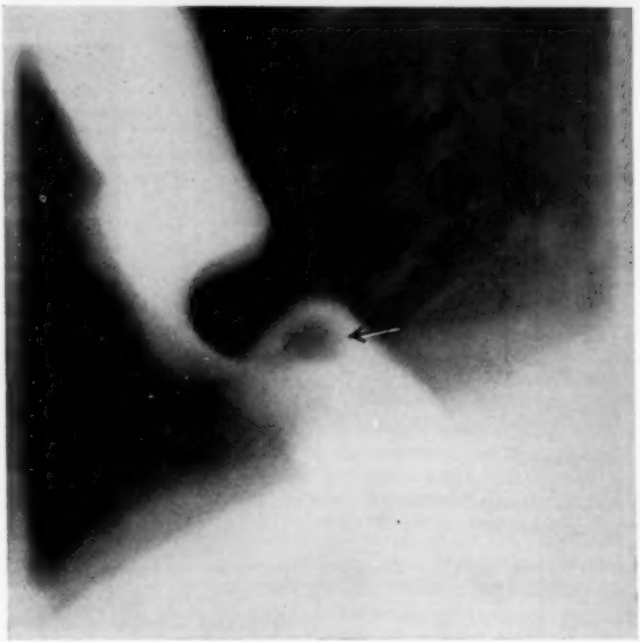

Fig. 10-b. Cricopharyngeus spasm. Smooth indentation of the posterior wall of barium column well shown. Note small collection of trapped air in esophagus just under contracted muscle (arrow).

Cricopharyngeus spasm is to be differentiated from neoplasms of the hypopharynx and other masses in the neck. In tumors of the posterior wall of the hypopharynx, the swelling and indentation are usually not restricted to the cricopharyngeus region. The width of the prevertebral space is thickened to various degrees, and the posterior wall of the

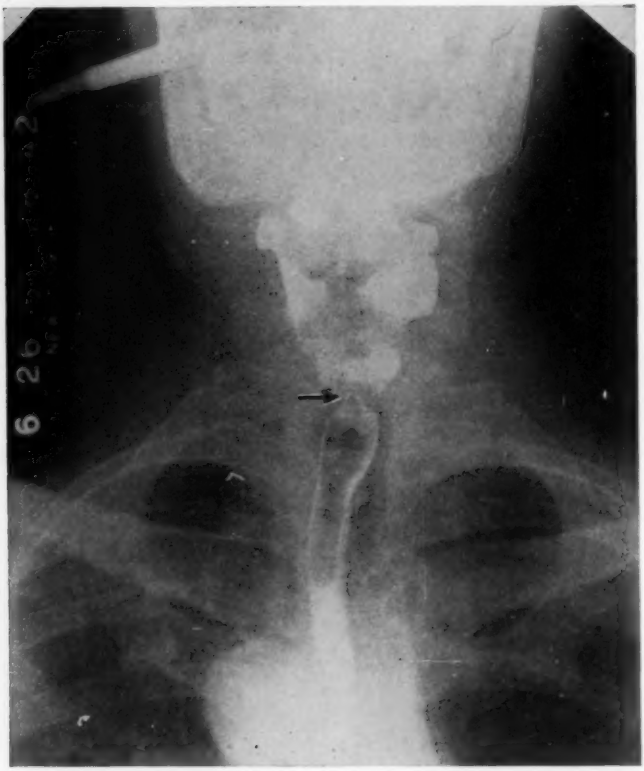

Fig. 11. Cricopharyngeus spasm. Frontal view. Note narrowed segment of barium column (arrow) between hypopharynx and cervical esophagus due to constriction by the spastic cricopharyngeus.

barium filled hypopharynx appears somewhate sinuous, due to the multiple indentations of the growing tumor. In other cases, there are indentations on the lateral and anterior surfaces of the hypopharynx by neoplasms situated in these areas.

SUMMARY.

1. The anatomy and physiology of the laryngopharynx are described in detail during the second stage of swallowing.

2. The value of a 9x9 inch film rapid changer permitting exposures up to a rate of two per second is demonstrated.

3. The mechanism of prevention of aspiration during swallowing is discussed.

4. A classification of abnormal mechanisms resulting in aspiration is presented.

5. Illustrative case reports are briefly presented.

6. The anatomy and action of the cricopharyngeus muscle during respiration and swallowing are discussed.

7. The radiographic appearance of cricopharyngeus spasm is described and its differential diagnosis from tumors of the hypopharynx and other portions of the neck detailed.

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The next joint annual meeting of the South Carolina Society of Ophthalmologists and Oto-laryngologists, and the North Carolina Eye, Ear, Nose, and Throat Society, will be held in Charleston, South Carolina, September 13-16, 1959. Headquarters will be the Francis Marion Hotel. Guest speakers will include Ophthalmologists: Dr. Caroll R. Mullen of Philadelphia, Pa.; Dr. G. Bonaccolto of New York, and Dr. Willis S. Knighton of New York; Oto-laryngologists: Dr. John Bordley of Baltimore, Md., Dr. Frederick R. Guilford of Houston, Tex., and Dr. Paul Holinger of Chicago, Ill.

For further details write Roderick Macdonald, M.D., Secretary and Treasurer, 330, East Main St., Rock Hill, S. Car.

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This bibliography was not included in THE LARYNGOSCOPE for May, 1959.

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Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting:

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Antonio Augusto de Almeida.
First Secretary: Dr. Alberto Galo.
Second Secretary: Dr. Alfredo Porto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. J. Penido Burnier, Dr. Guedes de Melo Filho and Dr. Roberto Franco do Amaral.
Meetings: Twice every month, first and third Thursdays, 8:30 P.M.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Sufie Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Rd., Halifax, Nova Scotia.
Meeting: Sheraton-Brock Hotel, Niagara Falls, Ontario, October 9-10, 1959.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. William F. Hubble, Decatur, Ill.
President-Elect: Dr. Charles D. Sneller, Peoria, Ill.
Vice-President: Dr. Edgar T. Blair, Springfield, Ill.
Delegate at Large: Dr. G. Leroy Porter, Urbana, Ill.
Secretary-Treasurer: Dr. Clarence A. Fleischli, Springfield, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago 3, Ill.
Vice-President: Dr. Maurice Snitman, 408 So. 5th Ave., Maywood, Ill.
Secretary-Treasurer: Dr. Fletcher Austin, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Fred D. Hollowell, Lamar Life Bldg., Jackson, Miss.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting:

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairmen in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Monterey 47, Mexico 7, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. David D. DeWeese, 1216 S. W. Yamhill St., Portland 5, Ore.
Secretary-Treasurer: Dr. Paul B. Myers, 223 Medical Dental Bldg., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Henry Thiele Restaurant, 23rd and W. Burnside, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. E. P. Fowler, Jr., 180 Fort Washington Ave., New York 32, New York.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2, Ky.
Meeting: Palmer House, Chicago, Ill., October 11, 1959.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118 - 9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President Dr. John J. O'Keefe.
Vice-President: Dr. Joseph P. Atkins.
Secretary: Dr. William A. Lell.
Executive Committee: Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, Dr. William A. Lell, Dr. William J. Hitschler, and Dr. Chevalier L. Jackson.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Macario G. Tan, 426 Evangelista, Manila, P. I.
Vice-President: Dr. Ariston G. Bautista, 460 Isaac Peral, Manila, P. I.
Secretary-Treasurer: Dr. Angel Enriquez, American Hospital, Aduana St., Manila, P. I.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 8101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

Meeting: University of Vienna, August 29 to September 1, 1960.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: Dr. F. T. Land, 13 Newton Place, Glasgow, C. 3.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow, C. 3.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Monterrey 47-201
Mexico 7, D. F.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela.
Treasurer: Dr. Benito Madariaga.
First Vocal: Dr. Rafael González.
Second Vocal: Dr. Juan Oberhauser.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr Gabriel Briceño Romero.
Vice-Presidente: Dr. Silvestre Rincón Fuenmayor.
Secretario General: Dr. Oscar Bustamante Miranda.
Tesorero: Dr. Arturo Marrero Gómez.
Vocales: Dr. Miguel Octavio Russa, Dr. Benjamin Briceño, Dr. Oscar Gonzalez Castillo.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Ivo Adolpho Kuhl.
Secretary: Dr. Decio Lisboa Castro.
Treasurer: Dr. Jorge Valentin.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. Maria Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Lt. Col. Stanley H. Bear, USAF (MC), USAF Hospital, Maxwell (Air University), Maxwell Air Force Base, Ala.
Secretary-Treasurer: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. F. R. Price, 118 Rutledge Ave., Charleston, S. C.
President-Elect: Dr. L. D. Lide, 161 W. Cheves St., Florence, S. C.
Vice-President: Dr. R. E. Livingstone, 1505 Main St., Newberry, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, 330 E. Main St., Rock Hill, S. C.
Meeting: Jointly with the N. C. Society of Eye, Ear, Nose and Throat. Next joint annual meeting of The South Carolina Society of Ophthalmology and Otolaryngology and the N. C. Eye, Ear, Nose and Throat Society at Charleston, S. C., Sept. 14, 15 and 16, 1959.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. V. Eugene Holcombe, Charleston, W. Va.
Chairman-Elect: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Vice-Chairman: Dr. George M. Haik, New Orleans, La.
Secretary: Dr. Mercer G. Lynch, New Orleans, La.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Va.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Va.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Nime K. Joseph, Wheeling, W. Va.
President-Elect: Dr. John A. B. Holt, Charleston, W. Va.
Vice-President: Dr. William K. Marple, Huntington, W. Va.
Secretary-Treasurer: Dr. Albert C. Esposito, Huntington, W. Va.
Director for Two Years: Dr. James T. Spencer, Charleston, W. Va.
Next Meeting: Greenbrier Hotel, White Sulphur Springs, W. Va., Aug. 20-23, 1959.